University of Alberta

The Alberta Fibrosis Program: Pulmonary Function in Workers Exposed to Coal, Silica, and Asbestos Dusts

by

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Abstract

The Alberta Fibrosis of the Lung Program was a legislated surveillance program of workers exposed to coal, silica, or asbestos dusts. Biennial examinations included a pulmonary function test (PFT) and chest x-ray. Out of 29237 individual records within the Fibrosis Program database, only 3895 subjects had at least one PFT and 2425 subjects had at least two PFTs plus adequate data (eg. age, sex, height) to perform multiple linear regression analyses. At the time of the last PFT, coal-exposed subjects had a 57 mL lower FEV_1 and 82 mL lower FVC as compared to subjects of other dust exposure types. Dust exposure experienced while enrolled in the Fibrosis Program did not appear to be related to clinically significant declines in pulmonary function, regardless of type of dust exposure. The overall crude prevalence of radiographic pneumoconiosis was 2.1%, with almost half of the pneumoconiosis cases identified at entry into the Program.

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Table of Contents

1.0 Introduction 1

1.1 Pneumoconiosis 1

1.2 Pneumoconiosis Surveillance 2

1.3 Pneumoconiosis in Canada 5

1.4 Alberta Fibrosis Program 6

- 1.4.1 History and Legislation 6
- 1.4.2 Policies and Procedures 8
- 1.4.3 Fibrosis Program Evaluations 11

1.5 Surveillance Outcome Measures 12

1.6 Study Rationale and Hypothesis 14

2.0 Dust-Specific Literature Reviews 15

2.1 Coal 15

- 2.1.1 Dose-Response 17
- 2.1.2. Greater Dose-Response Estimates and Age Effects 19
- 2.1.3 Survival Bias and Clinical Significance 23
- 2.1.4 Influence of Radiographic Pneumoconiosis 27
- 2.1.5 Influence of Smoking 28
- 2.1.6 Coal Summary 29

2.2 Silica 31

- 2.2.1 Radiographic Silicosis and Exposure-Response 32
- 2.2.2 Pulmonary Function and Exposure Response 33
  - 2.2.2.1 Miners 33
  - 2.2.2.2 Other Occupational Groups 38
- 2.2.3 Influence of Radiographic Pneumoconiosis 43
- 2.2.4 Influence of Smoking 46
- 2.2.5 Silica Summary 49

2.3 Asbestos 50

2.3.1 Historical Studies 51

2.3.2 Studies of Workers in Specific Industries 52

2.3.3 Studies of Workers in Specific Trades 56

2.3.4 Influence of Radiographic Pneumoconiosis and Pleural Changes 59

2.3.5 Influence of Smoking 63

2.3.6 Asbestos Summary 66

3.0 Methods 68

3.1 Fibrosis Program Database Content 68

3.2 Dependent Variables 69

3.2.1 FEV$_1$, FVC, FEV$_1$/FVC% 69

3.2.2 Percent-Predicted FEV$_1$ and FVC 69

3.3 Independent Variables 70

3.3.1 Date Values 70

3.3.2 Exposure Type 70

3.3.3 Duration of Exposure 71

3.3.4 Age 72

3.3.5 Sex 72

3.3.6 Height 72

3.3.7 Symptoms and Past Medical History 73

3.4 Subjects Available for Analysis 73

3.5 Analysis Steps 74

3.5.1 All Subjects (n = 29237) 75

3.5.2 Analyses of First PFT Outcomes for Subjects with at Least One PFT 75

3.5.2.1 Analyses Within Exposure Groups 75

3.5.2.2 Analyses Between Exposure Groups 75

3.5.3 Analyses of Last PFT Outcomes for Subjects with Two or More PFTs 76

3.5.3.1 Analyses Within Exposure Groups 76

3.5.3.2 Analyses Between Exposure Groups 77

3.5.4 Analyses of Last PFT Outcomes for Subjects with Two or More PFTs and Ten or More Years Exposure Duration 77

3.5.5 X-Ray Analyses 77

3.5.6 Smoking Analyses 78

3.6 Statistical methods 79

3.7 Ethical Approval 80

4.0 Results 81

4.1 All subjects (n = 29237) 81

4.1.1 Enrolment 81

4.1.2 Missing Data 81

4.1.3 Industry Representation 81

4.2 Analyses of First PFT Outcomes for Subjects with at Least One PFT 82

4.2.1 Descriptive comparisons 82

4.2.2 Simple Linear Regression 83

4.2.3 Within Exposure Group Analyses 83

4.2.4 Between Exposure Group Analyses 84

4.2.5 Between Exposure Group Analyses, No Previous Dust Exposure 85

4.2.6 Analysis Summary of First PFT Outcomes for Subjects with at Least One PFT 85

4.3 Analyses of Last PFT Outcomes for Subjects with Two or More PFTs 86

4.3.1 Descriptive Comparisons 86

4.3.2 Simple Linear Regression 87

4.3.3 Within Exposure Group Analyses 88

4.3.4 Within Exposure Group Analyses, No Previous Dust Exposure 89

4.3.5 Between Exposure Group Analyses 89

4.3.6 Between Exposure Group Analyses, No Previous Dust Exposure 90

4.3.7 Analysis Summary of Last PFT Outcomes for Subjects with Two or More PFTs 90

4.4 Analyses of last PFT Outcomes for Subjects with Two or More PFTs and Ten or More Years Exposure Duration 91

4.4.1 Descriptive Statistics 91

4.4.2 Within Exposure Group Analyses 92

4.4.3 Within Exposure Group Analyses, No Previous Dust Exposure 93

4.4.4 Between Exposure Group Analyses 94

4.4.5 Between Exposure Group Analyses, No Previous Dust Exposure 94

4.4.6 Analysis Summary of Last PFT Outcomes for Subjects with Two or More PFTs and Ten or More Years Exposure Duration 95

4.5 X-Ray Analyses 96

4.5.1 Descriptive Statistics 96

4.5.2 Pneumoconiosis and Pulmonary Function 97

4.5.3 Pneumoconiosis and Pulmonary Function, Regression Analyses 98

4.6 Smoking Analyses 100

4.6.1 Descriptive Comparisons 100

4.6.2 Regression Analyses, First PFT 100

4.6.3 Regression Analyses, Last PFT 101

5.0 Discussion 102

5.1 Main Findings, Within Exposure Group Analyses 102

5.2 Main Findings, Between Exposure Group Analyses 103

5.3 Main Findings - X-ray Analyses 106

5.4 Study Limitations 108

5.4.1 Selection 108

5.4.2 Exposure 110

5.4.3 Confounding 112

5.4.4 Outcome 113

5.4.5 Analysis 115

5.5 The Alberta Fibrosis Program 116

5.6 Summary 117

6.0 Tables 118

7.0 Figures 168

8.0 Bibliography 180

9.0 Appendices 197

Appendix 1: International Labour Office Classification of Radiographs of Pneumoconioses 198

Appendix 2: Pulmonary Function Testing 201

Appendix 3: Regulations and Legislation Pertaining to the Alberta Fibrosis Program 204

Appendix 4: Forms Provided for Use in the Alberta Fibrosis Program 237

List of Tables

Table 1: Occupations listed in Alberta Regulations 186/66 and 375/71 requiring inclusion in the Fibrosis Program. 119

Table 2: Definitions of exposed workers requiring inclusion in the Fibrosis Program. 120

Table 3: 8-Hour Occupational Exposure Limits for asbestos, silica, and coal dusts. 121

Table 4: Data contained within the original Fibrosis Program data files. 122

Table 5: Industry category at time of enrolment into the Fibrosis Program. 123

Table 6: Descriptive comparison of subjects with and without PFT data. 124

Table 7: Descriptive comparisons between dust exposure groups for subjects with at least one PFT. 125

Table 8: Results of simple linear regression analyses on first PFT parameters. 126

Table 9: Results of simple linear regression analyses on first PFT parameters for previous dust exposure, within exposure group comparisons. 127

Table 10: Results of multiple linear regression analyses on first PFT parameters, within exposure group comparisons. 128

Table 11: Results of multiple linear regression analyses on first PFT percent-predicted FEV_1 and FVC, within exposure group comparisons. 129

Table 12: Results of multiple linear regression analyses on first PFT parameters, between exposure group comparisons. 130

Table 13: Results of multiple linear regression analyses on first PFT percent-predicted FEV_1 and FVC, between exposure group comparisons. 131

Table 14: Comparison of mean first PFT percent-predicted FEV_1 and FVC between exposure groups. 132

Table 15: Descriptive comparisons of subjects with PFT data: subjects with and without two or more PFTs. 133

Table 16: Descriptive comparisons between dust exposure groups for subjects with two or more PFTs. 134

Table 17: PFT comparisons between dust exposure groups for subjects with two or more PFTs. 135

Table 18: Results of simple linear regression analyses on last PFT parameters. 136

Table 19: Results of simple linear regression analyses on last PFT parameters for exposure duration, within exposure group comparisons. 137

Table 20: Results of multiple linear regression analyses on last PFT parameters, within exposure group comparisons. 138

Table 21: Results of multiple linear regression analyses on last PFT percent-predicted FEV$_1$ and FVC, within exposure group comparisons. 139

Table 22: Results of multiple linear regression analyses on last PFT parameters, between exposure group comparisons. 140

Table 23: Results of multiple linear regression analyses on last PFT percent-predicted FEV$_1$ and FVC, between exposure group comparisons. 141

Table 24: Comparison of mean last PFT percent-predicted FEV$_1$ and FVC between exposure groups. 142

Table 25: Descriptive comparisons of subjects with two or more PFTs: subjects with ten or more years exposure duration versus subjects with less than ten years exposure duration. 143

Table 26: Descriptive comparisons between dust exposure groups for subjects with two or more PFTs: subjects with ten or more years exposure duration versus subjects with less than ten years exposure duration. 144

Table 27: Descriptive percent-predicted PFT value comparisons between dust exposure groups for subjects with two or more PFTs: subjects with ten or more years exposure duration versus subjects with less than ten years exposure duration. 145

Table 28: Results of simple linear regression analyses on last PFT parameters for exposure duration, within exposure group comparisons for subjects with ten or more years exposure duration. 146

Table 29: Results of multiple linear regression analyses on last PFT parameters, within exposure group comparisons for subjects with ten or more years exposure duration. 147

Table 30: Results of multiple linear regression analyses on last PFT percent-predicted FEV$_1$ and FVC, within exposure group comparisons for subjects with ten or more years exposure duration. 148

Table 31: Results of multiple linear regression analyses on last PFT parameters, between exposure group comparisons for subjects with ten or more years exposure duration. 149

Table 32: Results of multiple linear regression analyses on last PFT percent-predicted FEV$_1$ and FVC, between exposure group comparisons for subjects with ten or more years exposure duration. 150

Table 33: Comparison of mean last PFT percent-predicted FEV$_1$ and FVC between exposure groups for subjects with ten or more years exposure duration. 151

Table 34: Chest radiograph status across dust exposure groups. 152

Table 35: Chest radiograph status across PFT groups. 153

Table 36: Subject frequencies for maximum ILO code and ILO code of the last x-ray in the Fibrosis Program. 154

Table 37: Subject frequencies for duration of previous dust exposure prior to identification of pneumoconiosis, divided by exposure type. 155

Table 38: Mean first PFT parameters as a function of x-ray status for all subjects with at least one PFT. 156

Table 39: Mean first PFT parameters (A) and last PFT parameters (B) as a function of x-ray status for all subjects with two or more PFTs. 157

Table 40: Mean ages, PFT dates, and duration between the first and last PFT as a function of x-ray status for all subjects with two or more PFTs. 158

Table 41: Smoking status for subjects with PFT data, all exposure groups combined. 159

Table 42: Pulmonary function parameters as a function of smoking status, within exposure group comparisons for subjects with at least one PFT. 160

Table 43: Pulmonary function parameters as a function of smoking status, within exposure group comparisons for subjects with at two or more PFTs. 161

Table 44: Results of simple linear regression analyses on first PFT parameters for smoking, within exposure group comparisons. 162

Table 45: Results of multiple linear regression analyses (that included smoking as a variable) on first PFT parameters, within exposure group comparisons. 163

Table 46: Results of multiple linear regression analyses (with smoking removed from the regression model) on first PFT parameters, within exposure group comparisons. 164

Table 47: Results of simple linear regression analyses on last PFT parameters for smoking, within exposure group comparisons. 165

Table 48: Results of multiple linear regression analyses (that included smoking as a variable) on last PFT parameters, within exposure group comparisons. 166

Table 49: Results of multiple linear regression analyses (with smoking removed from the regression model) on last PFT parameters, within exposure group comparisons. 167

List of Figures

Figure 1: Comparison of frequencies of "other dust" and "silica" exposure codes at first PFT. 169

Figure 2: Subject samples available for analysis. 170

Figure 3: Subject enrolment in the Fibrosis Program for all dust exposure types. 171

Figure 4: Year of enrolment in the Fibrosis Program for subjects with coal exposure. 172

Figure 5: Year of enrolment in the Fibrosis Program for subjects with silica/other dust exposure. 173

Figure 6: Year of enrolment in the Fibrosis Program for subjects with asbestos exposure. 174

Figure 7: Frequency of subjects with coal, silica/other dust, and asbestos exposure codes for each industry category, all PFT groups combined. 175

Figure 8: Date of pneumoconiosis identification, all dust types combined. 176

Figure 9: Subject age at identification of pneumoconiosis for all subjects with incident pneumoconiosis. 177

Figure 10: Subject age at identification of pneumoconiosis for all subjects with pre-existing pneumoconiosis. 178

Figure 11: Comparison of PFT variability. 179

1.0 Introduction

1.1 Pneumoconiosis

Pneumoconiosis is a chronic lung disease defined as "the accumulation of dust in the lungs and the tissue reactions to its presence" (Cohen et al., 2002). Historically, the three most common pneumoconioses are those that result from the chronic inhalation of coal, silica, or asbestos dusts: coal workers' pneumoconiosis (CWP), silicosis, and asbestosis, respectively.

Although the pathophysiology of each of these pneumoconioses is unique, all three share similar features. Inhaled dust particles that are small enough to be respirable (ie reach the level of the respiratory bronchioles) accumulate in lung tissue if they overwhelm natural clearance mechanisms. The dust presence attracts local immune cells, alveolar macrophages, which act to try and remove the dust particles and respond to any tissue damage that has occurred secondary to the dust. The activation of these macrophages results in the release of reactive oxygen species, cytokines, and other inflammatory mediators, causing local tissue damage and the attraction of additional immune cells. Over time, this inflammatory response becomes chronic, resulting in fibrosis and local tissue remodelling. This fibrotic process progresses slowly over time with cumulative dust exposure. Eventually, the inflammatory changes of pneumoconiosis can lead to structural changes that can be visualized on a chest radiograph. As well, pneumoconiosis can result in pulmonary function impairment and respiratory symptoms (Banks et al., 2005; Brodkin et al., 2005; Petsonk et al., 2005). Further discussion regarding the pulmonary effects of exposure to coal, silica, and asbestos dusts is provided in sections 2.1, 2.2, and 2.3, respectively.

CWP, silicosis, and asbestosis are occupational lung diseases that are completely preventable if there is effective control of workers' exposure to occupational dusts (Bang et al., 1999). Because the natural history of these pneumoconioses is a slow progression over many years of chronic exposure, the potential exists to detect disease at an early stage, when removal from or reduction of exposure can limit disease progression. Subsequently, workers exposed to asbestos, silica, or coal dusts have frequently been the subjects of health surveillance, defined as "the ongoing collection, analysis, and timely reporting of health data for purposes relevant to disease prevention" (Wagner et al., 1993).

These surveillance programs typically focus on health outcomes related to pneumoconiosis, such as characteristic changes on chest radiography, declines in pulmonary function, respiratory symptoms, or some combination of the three. Perhaps the most widely used surveillance tool has been the chest radiograph, using a classification scheme devised by the International Labour Office (described in more detail in Appendix 1; details on pulmonary function testing can be found in Appendix 2). Chest radiographs have been favoured because they are relatively cheap, widely available, acceptable to workers, and there are standardized methods for radiograph production and interpretation (Wagner et al., 1993). Mortality has also commonly been used as an outcome measure to monitor pneumoconiosis trends.

1.2 Pneumoconiosis Surveillance

In the United States, the Centers for Disease Control and Prevention (CDCP) performs national surveillance for work-related lung disease. A variety of information sources are utilized, such as death certificates, reporting systems, industry-specific surveillance, and occupational clinic disease surveillance databases. The results of this surveillance are published regularly by the National Institute for Occupational Safety and Health (NIOSH), with the most recent report published in 2003 (NIOSH, 2003).

With respect to mortality (CWP, silicosis, or asbestosis listed as an underlying or contributory cause of death on a death certificate), this national surveillance program has observed that over time, deaths due to CWP or silicosis has declined while deaths due to asbestosis have increased in the United States. For CWP, age-adjusted mortality rates per million population declined steadily from a peak of 20 in 1972 to 4.71 in 1999. Similarly to CWP, silicosis mortality has also declined, from an age-adjusted mortality rate of 9 per million population in 1968 to 0.87 per million population in 1999 (NIOSH, 2003). A subsequent report published by CDCP analyzed data from the NIOSH National Occupational Respiratory Mortality System (NORMS) from 1968 to 2002, and found that silicosis was listed as an underlying or contributory cause of death for 16305 individuals out of 74 million death certificates examined (CDC, 2005). In contrast to declining mortality rates for CWP and silicosis, the NIOSH report noted that deaths due to asbestosis are on the rise: the age-adjusted mortality rate for asbestosis was 0.5 per million population in 1968, but has risen steadily up to 5.93 per million population in 1999. The number of deaths due to asbestosis in the United States was 1265 in 1999 (NIOSH, 2003). The reduction in CWP and silicosis mortality has been attributed to enforcement and compliance of dust control measures, although another important contributing factor for the decline in silicosis deaths has been the decline in employment in heavy industries where silica exposures are prevalent (Bang et al., 1999; CDC, 2005). Nevertheless, despite improved exposure controls, new CWP and silicosis cases continue to occur in the United States, even among young workers (CDC, 1998; CDC, 2003; CDC, 2005). It has been suggested that the increasing mortality for asbestosis could be related to the long latency of this disease and continuous long-term exposure, and it has been projected that deaths due to asbestosis will peak in 1999 and decline thereafter (Bang et al., 2005).

Workers exposed to coal dust are predominantly those involved in coal mining. This industry homogeneity has enabled relatively comprehensive industry-wide surveillance programs of miners. In the United States, national surveillance for

CWP is performed through the Coal Workers' X-Ray Surveillance Program (CWXSP), which is overseen by NIOSH. This is part of a lung disease prevention program mandated by the Federal Coal Mine Health and Safety Act of 1969. Under the CWXSP, companies offer underground coal miners a chest radiograph at first employment and every five years thereafter. Similarly to trends in mortality, the CWXSP has reflected a steady decline in the prevalence of CWP detected by x-ray among coal miners: among coal-miners with more than 25 years tenure, prevalence of CWP declined from 28% in 1970-1973 to 4% in 1997-1999 (NIOSH, 2003). In terms of overall CWP prevalence among miners, examination of over 250 000 chest radiographs collected from 1970 to 1986 demonstrated a consistent decline in overall CWP prevalence, from 10.5% to 1.6% (Althouse et al., 1992). This steady decline in CWP prevalence continued through to 1999 (CDC, 2003). Nevertheless, it has been noted that CWP continues to occur among miners employed only after 1970, when stricter coal dust exposure regulations came into effect (CDC, 2003).

However, several limitations to the database have been identified, many of which may have led to an underestimate of the true prevalence of CWP. These include miner participation (which has declined from 50% to less than 30% since 1970), over-representation of newly employed miners in later years of the program, and the possibility that workers with ill health are under-represented in multiple cross-sectional surveys (Attfield et al., 1992a, CDC, 2003, NIOSH, 2003). X-ray reader variability and changes in x-ray classification criteria may also affect interpretation of prevalence trends over time. A recent re-evaluation of x-rays collected over time using standardized criteria and a single team of 3 x-ray readers confirmed that CWP prevalence has declined steadily since the early 1970s. However, at each CWXSP survey round, the re-evaluation readings revealed a higher prevalence at each round. For example, in Round 1 (1969 to 1971), the summary prevalence of CWP as determined by the original readers was 4.5%, versus 7.3% for the re-evaluation, while in Round 4 (1985 to 1988), the corresponding values were 1.3% and 2.1% (Goodwin et al., 1998).

The declining trend in CWP has been noted in other countries as well. In Germany, 1369 coal miners who started underground work at two large collieries between 1974 and 1979 have undergone chest x-rays every two years. From 1974 to 1998, no cases of CWP with ILO category greater than 0/1 were identified in this worker cohort with an average of 15 years work underground. This contrasted sharply with the 5% of miners who developed CWP graded as > 1/1 between 1953 and 1973, after 15 years of underground work (Morfeld et al., 2002). In the United Kingdom, chest radiograph surveys of workers at 15 collieries have revealed that CWP prevalence fell from 12% in 1959 to 1963 down to 0.2% in 1994 to 1997 (Scarisbrick et al., 2002). However, it was also noted that CWP prevalence increased slightly to 0.8% in 1998-2000. The incidence of new cases also increased from 1.4 per 1000 individuals x-rayed in 1994 to 1997 to 6.9 per 1000 individuals x-rayed in 1998 to 2000. It has been noted that there has been the trend in recent years for miners to work longer hours than the standard working week (Kenny et al., 2002). Because statutory dust limits are set based on exposure-response relationships, longer than usual work hours would result in higher cumulative dust exposures, even though dust levels may be at "acceptable" levels (Kenny et al., 2002; Scarisbrick et al., 2002).

In contrast to coal mining, industries and trades that could entail exposure to silica dust are far more numerous and varied, making industry-specific surveillance challenging. In the United States, a small number of silicosis state-based surveillance systems exist (NIOSH, 2003). For example, a surveillance system in Michigan for silicosis has relied on a number of information sources to identify silicosis cases, including hospital reports, physician reports, death certificates, and claims awarded by an industry compensation fund. Silicosis was identified if a case had a history of silica exposure plus either a chest x-ray of ILO 1/0 or greater or a biopsy report of the characteristic silicotic nodule. The surveillance system identified 577 silicosis cases from 1985 to 1995, with 61% of these coming from hospital reports. Based on this surveillance program, it was estimated that the overall annual incidence for silicosis among black men and white men 40 years of age or older was 14.3 cases and 2.1 cases per 100 000, respectively (Rosenman et al., 1997).

However, it has been estimated that such surveillance schemes for silicosis greatly underestimate the true burden of new silicosis cases and silicosis deaths. Rosenman et al. (2003) extrapolated from the Michigan state-based surveillance scheme to estimate national rates of silicosis. Although NIOSH identified 2787 deaths in the United States due to silicosis from 1987 to 1996, it was estimated that the total number of newly recognized silicosis cases (living and deceased) during the same time period ranged from 36140 to 73179. Silicosis may be under-recognized and therefore not included on death certificates; a review of hospital records from decedents who worked in silica-exposed industries and whose cause of death was tuberculosis, cor pulmonale, or chronic obstructive lung disease found that 8.5% of cases had radiographic silicosis that had not been previously identified and documented on a death certificate (Goodwin et al., 2003). As well, employer-based surveillance programs may only cover part of the workforce (small businesses especially may not be included in such programs), employers may not be aware of chronic diseases such as silicosis that may only be diagnosed years after employment has ceased, and a substantial proportion of workers with silicosis may never apply for workers' compensation (Rosenman et al., 2003). Ultimately, the true number of current cases of silicosis and silica-related disease in the United States is unknown (NIOSH, 2003).

Large-scale surveillance for asbestosis has typically been geared towards specific trades through voluntary screening of active and retired union members. These surveys likely overestimate the true prevalence of asbestosis among active workers since participation is usually limited to union members who have at least 20 years experience in the trade, and workers who are unwell may be more inclined to participate. Selikoff et al. (1991) studied 1016 sheet metal workers who had at least 35 years in the trade and who attended examinations in 7 cities in the United States and Canada in 1986 and 1987, and found that 47% had radiographic asbestosis. A larger study screened almost 10000 sheet metal workers with a mean employment duration of 33 years at 56 U.S. facilities and 5 Canadian facilities starting in 1986; just over 12% of all screened workers had radiographic asbestosis (Welch et al., 1994). An earlier study evaluated 2611 asbestos insulators in 19 cities in the U.S. and Canada, and 60% of subjects had radiographic asbestosis (Miller et al., 1992).

1.3 Pneumoconiosis in Canada

In Canada, there is no systematic national surveillance program for pneumoconioses. As well, there is no systematic method of enumerating the population of workers exposed to pneumoconiosis-causing dusts in either primary or secondary industries. Without knowledge of the population at risk of developing pneumoconiosis it is impossible to accurately estimate the incidence and prevalence of pneumoconiosis in Canada (Ostiguy, 1979). Some indication of the burden of pneumoconiosis in Canada was provided in a Canadian Task Force report published in 1979. According to this report, two new cases of silicosis were identified each year per 1000 men employed in dusty jobs in Ontario between 1965 and 1976. In Manitoba in 1977, the prevalence of radiographic pneumoconiosis was 6.2 per 1000 employed men. In Quebec in 1976, the prevalence of asbestosis was 6.9% among 6875 employees of the asbestos mining industry (Ostiguy, 1979). Using data from provincial Worker's Compensation Boards, between 1965 and 1976 in Canada, there were 1235, 1035, and 877 compensated cases for CWP, silicosis, and asbestosis, respectively. However, data derived from Workers' Compensation awards are likely an underestimate of pneumoconiosis burden, as some affected individuals may not seek compensation and the diagnosis may not be recognized in normal clinical practice. Additionally, compensation awards "reflect more closely changes in social factors and legislation in regard to pneumoconiosis than changes in incidence and prevalence" (Ostiguy, 1979).

The number of deaths in Canada attributable to pneumoconiosis between 1965 and 1974 was 813. However, it was not known in how many cases pneumoconiosis was a contributory cause of death (Ostiguy, 1979).

In Ontario, a government surveillance program for workers in dusty industries has existed since the 1920's, relying on chest radiographs taken every two years to identify workers with silicosis. Out of 68701 workers in the surveillance database who were first exposed to silica dust in 1950 or later and still employed in 1979, 283 silicosis cases were identified (Finkelstein, 1994). Of those cases, 211 had been identified after 1979, with 11 to 26 new cases identified annually (Finkelstein, 1995). The detection rate increased with time from first exposure: less than 2 new cases were identified per 10000 examinations during the first two decades from first exposure, rising to 20-40 new cases per 10000 examinations after more than 27 years from exposure (Finkelstein, 1995).

In Alberta, reports have been published on coal miners and asbestos-exposed workers. At the time of a 1981 report, there was one underground and 17 surface coal mines in operation in Alberta, employing 400 underground miners and 1600 surface miners (Kaegi, 1981). Although the total workforce number had been relatively stable in the preceding decade at 1600 to 2300 total workers, the industry had a high turnover rate. Based on estimates from underground miner union representatives, only 10% of the workforce had 10 or more years of mining experience and only 3% had 15 years or more experience. Based on data from the Alberta Fibrosis Program and the Alberta Workers' Compensation Board, the prevalence of Coal Workers' Pneumoconiosis was 0.5% in the late 1970's; however, it was noted that Coal Workers' Pneumoconiosis rarely became radiologically evident before 10 to 15 years of exposure and only a small proportion of Alberta's coal miners had sufficiently long exposure to allow the development of the disease (Kaegi, 1981). With regards to coal dust exposure, periodic dust surveys of underground mines in the 1970's revealed that the average and median values for respirable coal dust at the coalface were 6 mg/m^3 and 5 mg/m^3, respectively. For coal preparation plant workers, dust concentrations from personal samples were typically 3 to 4 mg/m^3 (Kaegi, 1981).

For asbestos-exposed workers in Alberta, union officials and company representatives were surveyed in an effort to determine the number of workers potentially exposed to asbestos in 1977 (Kaegi, 1978). Potentially exposed occupational groups included asbestos distribution, asphalt production, cement, putty, and caulking compound manufacture, plastering, stuccoing, and drywalling, demolition, industrial insulation, residential insulation, maintenance, brake application, plumbers and pipefitters, and laundry service for asbestos workers' clothing. It was estimated that in 1977, between 2000 and 3000 active workers used asbestos or asbestos products in Alberta, and an additional 3000 workers had potentially been exposed to asbestos in the past (Kaegi, 1978). Based on Alberta Workers' Compensation Board data from 1968 to 1977, 19 men were diagnosed with asbestosis. For the reasons mentioned above, the compensation figure was likely an underestimate of the true number of asbestosis cases (Kaegi, 1978). Two small studies of boilermakers and plumbers and pipefitters in the Edmonton area have recently been published, and radiographic asbestosis was not observed in any cases (Hessel et al., 1998a, Hessel et al., 1998b).

1.4 Alberta Fibrosis Program

1.4.1. History and Legislation

Similarly to Ontario, the Government of Alberta has also operated a surveillance program for dust-exposed workers, referred to as the Fibrosis of the Lung

Program, or simply the Fibrosis Program. The aim of the Fibrosis Program was "to prevent disability due to dust-induced diseases of the lung". There were three stated objectives of the Program, which were:

- to identify dust induced diseases of the lung at the earliest stage in the population at risk in Alberta
- to promptly inform the worker and any person designated by the worker of any abnormal findings detected as a result of investigations performed under the Fibrosis Program
- to develop and maintain an efficient and effective organization for the Fibrosis Program

The Program's origins date back to the early 1960's, when Dr. Donovan Ross, the Minister of Health and Development, identified the need for an occupational health program in Alberta. Dr. Ross elicited the help of Dr. Herman Siemens, who in 1964 travelled throughout Alberta in a truck housing a mobile x-ray unit and pulmonary function testing equipment, in order to survey workers in dusty trades. Based on the findings of this 1964 survey, Dr. Ross established the Division of Industrial Hygiene within Alberta Health. The first item of legislation from this new Division was the Alberta Fibrosis Program (personal communication, Dr. Rodney May, 2003).

The Alberta Fibrosis Program was officially created by an amendment to the 1957 Public Health Act, Alberta Regulation 186/66, and came into effect on July 1, 1966 (copies of this amendment and all relevant regulations pertaining to the Alberta Fibrosis Program can be found in Appendix 3). The "Regulations Respecting the Protection of Persons from Fibrosis of the Lungs" stated that

> Every person who is engaged in any occupation where he is or may be exposed to the inhalation of any substance which may produce fibrosis of the lungs shall submit not less than once every two years to an examination to include a 14" x 17" chest x-ray and a pulmonary function test.

This requirement applied to any person involved in any occupation that was listed as part of the amendment. The initial examination was to be performed within two years of commencing employment. All examinations were to be performed by officials designated by the Provincial Board of Health and costs were to be borne by the Department of Public Health. Chest x-rays, pulmonary function test results, and all medical reports and supporting documentation were to be submitted to the Division of Occupational Health and Safety, which was responsible for filing and storage of all Fibrosis Program records.

In 1971, the initial Fibrosis Program Regulation was amended (AB Reg 375/71). Additional occupations were added to the original list (the complete list of occupations is included in Table 1) and the 1971 amendment also specified that the pulmonary function test would include measurement of FVC and FEV_1 (see Appendix 2). Perhaps the most significant change to the Regulation was that the responsibility for the costs of the biennial examinations shifted from the Department of Public Health to the employer.

From its inception until 1976, the Fibrosis Program was the responsibility of the Director of the Division of Industrial Health Services of the Department of Public Health. On December 1, 1976, The Alberta Occupational Health and Safety Act became law, which changed the administrative responsibility for the Fibrosis Program. This responsibility was now placed under the Director of Medical Services in the Division of Occupational Health and Safety.

In 1982 and 1983, the Alberta Fibrosis Program Regulation and its amendments were replaced by three separate Regulations: the Asbestos (AB Reg 7/82), Silica (AB Reg 9/82) and Coal Dust (AB Reg 243/83) Regulations. These new regulations outlined specific inclusion criteria for Fibrosis Program participants that were based on specific dust exposure levels defined by the Chemical Hazards Regulation (AB Reg 242/83), as opposed to simply membership in a particular occupation. The definitions of exposed workers who were to be included in the Fibrosis Program and the Alberta Occupation Exposure Limits that existed at the time of the Fibrosis Program are provided in Tables 2 and 3, respectively. Requirements for additional information, such as medical history, that was to be included with each biennial examination were also specified.

In 1988, an amendment to the Chemical Hazards Regulation (AB Reg 393/88) reduced the Occupational Exposure Limits for “all other asbestos fibres” from 2 fibres/cm^3 of air to 0.5 fibres/cm^3 of air, which would have slightly altered the inclusion criteria for asbestos-exposed workers in the Fibrosis Program. In 1997, the Asbestos, Silica, and Coal Dust Regulations were repealed (AB Reg 169/97). Under this amendment, dust exposed workers were still required to undergo biennial medical examinations, but employers became responsible for the storage of all health assessment information. Because reporting of medical examination results to a central government office no longer occurred, the surveillance aspect of the Alberta Fibrosis Program ceased with this amendment.

1.4.2. Policies and Procedures

Although the Fibrosis Program was legislated in 1966, it was not until 1981 that formal documentation regarding standardized procedures was made available. The first document, “Policy and Procedures Manual for the Fibrosis of the Lung Program”, was released in January 1981, and the second document, “Fibrosis Program Information Manual”, was released in August 1981. The following information regarding Fibrosis Program procedures was obtained from these two references.

Employers were instructed to check the Fibrosis Program Regulation in order to determine if any of their occupations were listed among those required to participate. In general, whether or not an employer was selected to participate in the Program was determined by the employer's perception of exposure (Alleyne, 1988). If an occupation was not listed but there were dust exposure concerns, a dust sampling survey was to be performed and if any doubt, the Medical Services Branch was to be contacted. Once a decision had been made to enrol workers in the Fibrosis Program, the Regional Senior Medical Consultant of the Medical Services Branch of Workers' Health, Safety and Compensation was to be informed of the company's intention to implement the new program, as well as information regarding the company's name, address, type of hazard, number of exposed employees, the date the program was to be started, and whether a site survey had been performed and the results.

Workers were initially identified as "exposed" based on their occupation, and after 1982, based on their duration of exposure to dust at a certain percentage of that dust's Occupational Exposure Limit (see Table 3). Specific measurements of worker dust exposures or information regarding the use of respiratory protective equipment were not collected as part of the Fibrosis Program. Using the worker's exposure history, job title, and employer information that were provided on each examination's assessment form, a single digit exposure code was assigned to each worker at each examination. This code represented exposure to coal, silica, asbestos, other dust, or man made mineral fibre in the period of time that had elapsed since the previous examination for that worker. A single exposure code was assigned at each examination.

Several forms were used to collect information on workers participating in the Fibrosis Program (Appendix 4). The identification form contained sections for company and worker identification, smoking status, and date and results of the pulmonary function test and chest x-ray. A separate employment and exposure history form requested previous job title, work type, employment duration, and employer name, as well as the onset and duration of exposure to specific dusts: asbestos, coal, silica, fibreglass, other dust. A new form was created in 1992 that consolidated the above information onto a single one-page form. In addition to the above information, sections on chest symptoms and past illnesses were added, as well as more detailed questions on smoking history and fitness to wear a respirator. All x-ray plates, PFT spirograms, assessment forms, and all other collected information were required to be forwarded to the main Alberta government office responsible for administering the Fibrosis Program. All data were stored manually and in the mid-1980's through the 1990's, a coding system was used to transfer the collected information into a computerized database.

At the commencement of the Fibrosis Program, workers were required to undergo an examination that included a 35 cm x 43 cm chest x-ray and a pulmonary function test (PFT), consisting of an FEV_1 and FVC, not less than every two years. With the introduction of the newer Asbestos, Silica, and Coal Dust

Regulations in 1982 and 1983, more explicit instructions were provided regarding the timing of examinations. For coal dust and silica-exposed workers, examinations were to take place not later than every 24 months for as long as the worker satisfied the definition of an exposed worker. For asbestos-exposed workers, examinations were to take place every 24 months for the first ten years of exposure, and every 12 months thereafter for as long as the worker satisfied the definition of an exposed worker. A worker could only be removed from the Fibrosis Program if the company no longer used any substances that could cause fibrosis of the lung or if the worker was no longer exposed to such substances and the worker's termination x-ray and PFT were normal. In the situation where the termination x-ray or PFT were abnormal, the worker was to continue in the Program.

The chest radiographs were assessed by community radiologists. The Senior Medical Consultant within the Alberta Occupational Health and Safety Division would review the radiologists' reports in order to determine if the chest x-ray was normal, demonstrated non-occupational disease, or demonstrated occupational disease. Chest radiographs were also coded according to the International Labour Office Classification of Radiographs of Pneumoconioses. During the life of the Fibrosis Program, different readers performed this coding task, some who were certified "B" readers and some who were not. Initially, the work of a "B" reader was supplemented by other consultant radiologists and radiology residents who worked on a fee for service basis. From the 1980's until 1992, a single consultant radiologist coded the chest radiographs. After 1992, due to a lack of interest by Alberta radiologists, sets of radiographs were sent on a monthly basis to a radiologist in Hamilton, Ontario to continue the process of ILO coding (personal communication, Dr. Rodney May, 2003).

The Fibrosis Program Information Manual published in 1981 also stipulated that PFTs were to be conducted by an individual who had passed an approved pulmonary function technician course. This stipulation was included because "in the past, the quality of pulmonary function tests has not been very good" (Fibrosis Program Information Manual, 1981). A 1982 report noted that of the submitted pulmonary function tests, 9.1% were identified as unacceptable for one of several reasons: less than three spirometry tracings, evidence of inadequate performance, poor expiratory effort, or a faulty machine (Alleyne, 1982). The requirement qualified pulmonary function technicians was later included in the Asbestos (AB Reg 7/82), Silica (AB Reg 9/82) and Coal Dust (AB Reg 243/83) Regulations in 1982 and 1983. Irrespective of these quality control measures, there is no way to confirm that either equipment or personnel met minimal standards. There is also no way of determining if workers were tested by the same personnel and on the same equipment at each subsequent examination.

1.4.3 Fibrosis Program Evaluations

Several shortcomings of the Fibrosis Program as a surveillance program have been noted in previous reports. One of these was the inability to accurately identify the population at risk: "an effective means for identifying every company where there may be workers exposed to asbestos, silica, or coal dust is not available and a reliable estimate of the number of Alberta workers who should be covered by the Fibrosis Program is not possible" (Alleyne, 1988).

Compliance with the program was also a major concern. It was not verified whether or not inclusion criteria were followed and the proportion of the Alberta working population at risk of fibrosis of the lung who were included in the Fibrosis Program was unknown (Alleyne, 1982; Alleyne, 1988). Among coal mining companies, comprehensive examinations were conducted at the time of hiring, as required, but periodic medical assessments thereafter were "infrequent, incomplete, or non-existent" (Kaegi, 1981). The same coal mining report also noted that the "Fibrosis Regulations were difficult to administer due to a constantly changing workforce and a lack of facilities to administer the required radiological and pulmonary function tests" (Kaegi, 1981). Very few asbestos-exposed workers had entered the Fibrosis Program prior to 1972 (Alleyne, 1982). This may have been due to an initial lack of asbestos-worker union support, as some unions resisted because of the mandatory nature of the Program and the access to personal information, such as smoking history (personal communication, Dr. Rodney May, 2003). However, this improved in subsequent years and in a later report, it was noted that 89% of the 1086 active members of the Heat and Frost Insulators Asbestos Workers Union participated in the Fibrosis Program (Alleyne, 1988).

An estimate of the level of compliance with the Program was determined by examining the submission of x-rays, which should have been done on a biennial basis. Based on the 13948 records on file by the end of 1979, there should have been 11181 subjects who submitted a second x-ray, but only 3855 (34.5%) did so. Only 19.6% of expected third x-rays, 11.3% of expected fourth x-rays, 5.4% of expected fifth x-rays, and 2.3% of expected sixth x-rays were submitted. This poor compliance was noted to be "disturbing" and "one of the major weak areas of the Fibrosis Program, and one which must be corrected if the program is to achieve its stated objective of identifying dust induced diseases of the lung at the earliest stages possible to prevent disability" (Alleyne, 1982). Poor compliance with the Fibrosis Program was also demonstrated when the names of 36 cases of asbestosis, accepted by the Workers' Compensation Board, were examined against the names of workers listed in the Fibrosis Program. Of the 36 claims, only four were found to be workers who had participated and had records in the Fibrosis Program (Alleyne et al., 1994).

The presence of pneumoconiosis as determined by chest radiographic interpretation was evaluated in a study of 15235 records (representing 11201 workers) which were submitted between January 1, 1981 and December 31, 1984 to the Fibrosis Program (Alleyne, 1988). During this time period, the same radiologist assigned an ILO grade to all submitted x-rays. A sample of 70 x-rays graded 1/1 or higher by this radiologist were also read by external readers in Ontario and the United States, and only 11 x-rays were graded as 1/1 or higher by the external readers. It was concluded that the Alberta reader generally assigned higher ILO grades, such that there was a high likelihood of false positive radiographic pneumoconiosis readings.

Of the x-rays reviewed, 28.5% (4344) were from asbestos workers, 19.4% (2955) were from silica workers, 27.8% (4239) were from coal workers, and 24.3% (3697) were from workers exposed to other dust. Eighty-three x-rays were classified as pneumoconiosis, and these films represented 72 individuals: 43 asbestosis, 16 silicosis, 11 coal workers' pneumoconiosis, and 2 non-specified pneumoconiosis. Eighteen of the 72 workers had pneumoconiosis at their first submission, suggesting that many of the workers had substantial dust exposure prior to enrolment in the Fibrosis Program (this was especially true for asbestosis). Thirty-eight workers with x-rays of 1/1 or greater had normal x-rays prior to 1981. Based on those workers who developed pneumoconiosis during the observation period, incidence rates per 1000 person years of observation were 4.0 for asbestos, 7.2 for silica, and 1.1 for coal dust. Using 11 201 workers as a denominator, four-year period prevalence was greatest for asbestosis at 13.6 per 1000 exposed workers, followed by silicosis at 10.4 cases and CWP at 3.4 cases per 1000 workers (Alleyne, 1988).

By 1994, of 27452 individual records contained within the Fibrosis Program database, 192 workers had pneumoconiosis (chest radiograph with an ILO grade of 1/1 or greater) (Alleyne et al., 1994).

1.5 Surveillance Outcome Measures

Most pneumoconiosis surveillance schemes have relied on chest radiography to identify cases of disease, due to reasons such as cost, acceptability, and standardized interpretation methods. However, in order for the changes of pneumoconiosis to be visible on a chest radiograph, the inflammatory lesions of the disease must be of a size and density to cause x-ray beam attenuation. As discussed further in 2.1, 2.2, and 2.3, a substantial degree of dust accumulation and subsequent inflammatory changes have already occurred by the time pneumoconiosis is evident on a chest radiograph. Therefore, the chest radiograph can be insensitive to early or moderate degrees of pneumoconiosis, thereby limiting its effectiveness as a screening tool (Wagner et al., 1993).

A normal chest radiograph does not exclude the presence of fibrotic changes consistent with pneumoconiosis. Tissue specimens obtained from biopsy or at autopsy from dust-exposed individuals with normal chest radiographs can reveal the presence of fibrosis (Hnizdo et al., 1993; Kipen et al., 1987, Rosenman et al., 1997). More sensitive imaging modalities, such as high resolution computed tomography (HRCT), can identify pneumoconiosis not seen on conventional chest radiographs (Lebedova et al., 2003; Neri et al., 1996; Soulat et al., 1999; Staples et al., 1989). For asbestosis, Ross (2003) estimates that the chest radiographic may be normal 10 to 15% of the time, yielding a sensitivity of 85 to 90%.

Although the ILO classification system (see Appendix 1) provides a standardized interpretation system, both intra- and inter-reader variability of radiograph interpretation has been a perennial concern (Henry, 2002). Reasons for this variability include reader experience, availability of standard classification films, and film quality (Henry, 2002). As well, many factors other than dust exposure can lead to mildly abnormal radiographic findings, such as radiographic technique, aging, obesity, smoking, and the presence of chronic obstructive pulmonary disease, thereby reducing the specificity of chest x-rays to detect pneumoconiosis. A review of articles that included subjects unexposed to dusts and use of the ILO system to classify radiographs observed that the prevalence of small opacities graded 1/0 or higher ranged from 0.21% to 11.7% across studies (Meyer et al., 1997). Because of these factors, it is estimated that among present-day asbestos-exposed workers, for example, the positive predictive value of chest radiograph alone is too low to diagnose asbestosis with confidence (Ross, 2003). A recent study that examined the concordance of radiograph interpretation among qualified readers observed that the agreement among readers was greater for normal radiographs than for ones that showed disease (Welch et al., 1998). Using an estimated population prevalence of pneumoconiosis of 10% and calculated positive and negative predictive values, the study results suggested that there would only be about a 50-50 chance that qualified readers would agree on the classification of an individual radiograph as positive for disease (Welch et al., 1998). However, although reader variation may be considerable, it rarely exceeds one classification category (Welch et al., 1998; Henry, 2002).

Because of these limitations of chest radiography, perhaps the most important outcome measure regarding worker health is the influence of dust exposure on pulmonary function, as opposed to radiographic changes per se. Although there is a good correlation between severity of radiographic changes and pulmonary function, several authors have suggested that the amount of cumulative dust exposure is the more important predictor of pulmonary impairment, as opposed to the degree of visible x-ray changes (Attfield et al., 1992b; Hnizdo, 1992; Irwig et al., 1978; Rogan et al., 1973; Soutar et al., 1986;). With respect to coal miners, Hurley et al. (2002) noted that impairment of lung function is independent of CWP, except insofar as both are related to cumulative dust exposure, and so an assessment of CWP impacts only will underestimate the overall burden of disease. Indeed, several studies of workers exposed to coal, silica, or asbestos dusts have demonstrated that exposure-related pulmonary function deficits can be observed in the absence of pneumoconiosis on standard radiographs (Attfield et al., 1992b; Carta et al., 1996; Erdinc et al., 2003; Harkin et al., 1996; Hertzberg et al., 2002;

Humerfelt et al., 1998; Kilburn et al., 1994; Lewis et al., 1996; Malmberg et al., 1993; Meijer et al., 2001; Miller et al., 1994; Neri et al., 1996; Staples et al., 1989; Soutar et al., 1986; Wang et al., 1997a; Wang et al., 2000a; Zuskin et al., 1998).

1.6 Study Rationale and Hypothesis

As noted above, previous evaluations of the Alberta Fibrosis Program have only focussed on chest radiographs as an outcome measure (Alleyne et at., 1982; Alleyne et at., 1988; Alleyne et at., 1994). Radiographic pneumoconiosis is graded according to the ILO profusion score, based on a 12-point ordinal scale. Such a gross measurement negates the discrimination of minor differences between subjects. Conversely, pulmonary function parameters such as FEV_1 and FVC are continuous variables, allowing for greater discrimination of changes and differences within and between subjects. Although subtle changes in pulmonary function may not be noticeable above expected variation at the individual level (Townsend, 2005; Wang et al., 2004), small systematic changes in pulmonary function across large groups can be detected at the population level. Therefore, if adverse effects of dust exposure have occurred in Alberta workers enrolled in the Fibrosis Program, analysis of pulmonary function would be far more sensitive to subtle effects than x-ray analyses. This is bolstered by the observation in several studies of pulmonary function changes in dust-exposed workers without radiographic pneumoconiosis, cited above.

To date, a large-scale evaluation of pulmonary function in the Fibrosis Program cohort has not been performed. The Alberta Fibrosis Program database offers a unique opportunity to compare longitudinal changes in pulmonary function parameters in a large cohort of workers exposed to different types of dust. In this manner it may be possible to determine if workers who are exposed to a particular type of dust suffer greater losses in pulmonary function than workers exposed to other types of dust. It is hypothesized that pulmonary function parameters (FEV_1, FVC, FEV_1/FVC) at last contact with the Fibrosis Program will not differ significantly between workers exposed to coal, silica, or asbestos dusts.

2.0 Dust-Specific Literature Reviews

2.1 Coal

Coal is predominantly a carbonaceous material, comprised of carbon, hydrogen, oxygen, sulphur, and trace elements. Mining for coal entails exposure not only to coal dust, but also to other mineral dusts during the cutting of rock during coal extraction. Dust particles generated from mining range in size from 1 to 50 μm in diameter, with coal face workers generally receiving the highest dust exposure (Attfield et al., 1996a).

Fine coal dust that is inhaled and overloads lung clearance mechanisms accumulates in lung macrophages at the level of the respiratory bronchioles. This deposition triggers the release of inflammatory mediators and reactive oxygen species, leading to tissue damage and remodelling. Dust-laden macrophages amass into dust macules, lesions that are 1 to 5 mm in size. At this stage, reticulin is present in the macules, but with little collagen. There is destruction and distension of adjacent alveolar walls, resulting in focal emphysema. As the disease advances, macules enlarge and coalesce, forming nodules. Nodules are much firmer than macules, show clear collagen deposition, and range in size from 2 to 10 mm (Attfield et al., 1996a; Petsonk et al., 2005).

These macules and nodules are the characteristic lesions of simple Coal Workers' Pneumoconiosis (CWP). The primary risk factor for CWP is the extent of exposure to coal mine dust. Coal rank (a factor related to the hardness and carbon content of the coal) is also a risk factor: CWP risk rises with increasing coal rank (Attfield et al., 1996a). Chest radiographs of miners with simple CWP often reveal small opacities, resulting from the attenuation of the x-ray beam by the inflammatory macules and nodules. The profusion of these small opacities seen on a chest radiograph correlates well with the size and number of pathologic lesions, and is the basis for the ILO classification (see Appendix 1). A substantial degree of dust deposition and subsequent inflammatory changes have to take place before CWP is evident on a chest radiograph (Attfield et al., 1996a).

With further progression of the disease, coalescence of nodules can occur, resulting in larger lesions (usually one to two centimetres in diameter or greater) comprised of dust particles, reticulin, collagen, and dust-engorged macrophages. These lesions typically appear in the upper lobes and are often associated with bronchitis and extensive emphysema. This stage of disease is referred to as complicated CWP or Progressive Massive Fibrosis (PMF). The factors leading to progression from simple to complicated CWP are not clearly understood, but excessive lung dust deposition is a necessary prerequisite (Attfield et al., 1996a; Petsonk et al., 2005).

The disease process of CWP can negatively affect pulmonary function. Bronchitic changes in the larger airways can increase resistance to airflow into and out of lungs. Airflow obstruction can also result from emphysematous destruction of elastic lung tissue at the levels of the bronchioles and alveoli. Dilation of small air spaces can lead to overinflation and gas trapping, while fibrotic lesions, particularly those associated with complicated CWP, can reduce the volume of air contained in the lungs, leading to a more restrictive lung function abnormality (Petsonk et al., 2005). FEV_1, FVC, and FEV_1/FVC all appear to decline roughly in parallel with increasing dust exposure, suggesting that coal dust exposure can lead to both an obstructive and restrictive effect on the lung (Attfield et al., 1996a).

The majority of studies that have examined the relationship between coal dust exposure and lung function have been derived from two large-scale research programs, Pneumoconiosis Field Research (PFR) in Great Britain and the National Study of Coal Workers' Pneumoconiosis (NSCWP) in the United States. The PFR program consisted of medical surveys conducted at five-year intervals. The initial survey from 1953 to 1958 included over 30000 coal miners from 25 collieries. Twenty-four of those collieries were included in the second (1958-1963) and third (1963-1968) surveys. For the fourth (1970-1973) and fifth (1973-1977) surveys, the number of surveyed collieries was reduced to 10. At each survey, a chest radiograph was obtained and information was collected regarding smoking habits, occupational history, and symptoms. Lung function measurements were included at the second survey and continued for subsequent surveys. For the ten-year period between the first and third surveys, extensive monitoring of respirable dust exposures took place. This monitoring continued in the 10 collieries that remained after the third survey. All of the original 24 collieries were included in a follow up survey conducted from 1974 to 1980 (Coggon et al., 1998; Soutar et al., 2004).

The NSCWP program in the United States was initiated after the passage of the Federal Coal Mine and Safety Act in 1969, which legislated that respirable coal dust exposures be less than 3 mg/m^3 starting in 1970, and less than 2 mg/m^3 by 1973 (Coggon et al., 1998; Seixas et al., 1992). Similarly to the PFR program, the NSCWP study consisted of surveys performed at five-year intervals: Round 1 (R1) in 1969-1971, R2 in 1972-1975, R3 in 1977-1981, and R4 in 1985-1988. The data collected at each survey for the 31 nationally distributed mines included occupational histories, symptoms, smoking habits, chest radiographs, and pulmonary function measurements. The major difference between the PFR study and the NSCWP study was the assessment of exposure to respirable coal dust: in the PFR study, dust samples were obtained for the purposes of epidemiological study whereas in the NSCWP study, samples were obtained for legal compliance purposes. Dust sampling obtained for compliance purposes may not have been representative of exposures on non-inspection workdays. For example, specific occupations within the mine were preferentially sampled, as opposed to a random sample of workers. As well, mine operators may have adjusted control measures during the time of the inspection in order to reduce dust levels and avoid citation by the inspectors. These biases, as well as the potential for other systematic errors from the compliance sampling protocol, may have led to great uncertainty in exposure-response estimates (Seixas et al., 1990).

Although the PFR and NSCWP surveys have been influential, they both had important limitations (Attfield et al., 1996b; Oxman et al., 1993; Stenton et al., 1993). Neither of the cohorts was truly representative of the coal-mining industry, as only large mines with long life expectancies were preferentially selected. Due to the cross-sectional nature of the studies, they may have underestimated exposure-response relationships because of the healthy worker effect, as workers who experienced adverse health effects from dust exposure may have died, left the industry, or moved to less dusty jobs within the industry, and subsequently would not have been included in the surveys. Longitudinal studies that used results from miners who participated in several surveys would have been affected by the same selection bias if only current miners were included; the exclusion of workers who left the mining industry during the observation period, some of whom may have had health problems, would likely underestimate the observed relationship between coal dust exposure and pulmonary function. The limits of the assessment of coal dust exposure in the NSCWP study has been mentioned, but even in the PFR study, the only exposure measured was respirable coal dust. Other workplace exposures that may have affected pulmonary function and symptoms (for example, other dusts such as silica, or diesel exhaust) were not accounted for, and so the extent to which the observed lung function changes may have been due to other coalmine exposures cannot be assessed.

2.1.1 Dose-Response

Despite the limitations cited above, the PFR and NSCWP surveys had the advantage of being able to study large numbers of workers. As such, these surveys have been instrumental in quantifying the relationship between coal dust exposure and decline in ventilatory function. One of the first attempts to quantify this relationship used data from the third PFR survey (Rogan et al., 1973). Subjects were 25 to 65 years old, had worked at the coalface for the first two surveys, and were still working underground at the time of the third survey. After exclusion of ex-smokers and subjects with progressive massive fibrosis (PMF), 3581 subjects remained. The average dust exposure for the group was 175 gram-hours per cubic meter (gh/m^3). Multiple regression analyses that included dust exposure, smoking, age, height, and weight as variables revealed that this average dust exposure was associated with a 105 mL loss of FEV$_1$, equivalent to a decline in FEV$_1$ of 0.60 mL per gh/m^3. From this cross-sectional study, it was estimated that a coal dust exposure of 4 mg/m^3 at the coalface (the British coal dust standard at the time) over a 35-year career, equivalent to 240 gh/m^3, would be associated with an average FEV$_1$ decline of 150 mL (Rogan et al., 1973).

Because of exposure estimate concerns, this same group of workers was re-analyzed utilizing numerical corrections to the pre-survey estimates of exposure (Marine et al., 1988). Regression estimates of the average effects of coal dust, after adjusting for age, height, and weight, were somewhat greater than for Rogan et al. (1973), with FEV_1 losses of approximately 100 mL for every 100 gh/m^3 of dust exposure, equivalent to a decline in FEV_1 of 1.0 mL per gh/m^3.

Complementing these estimates were the results from the largest cross-sectional study that used data from the NSCWP program. Subjects were 7139 white men older than age 25 who took part in the first survey (R1), conducted between 1969-1971 (Attfield et al., 1992b). Exposure information was derived from compliance samples obtained from 1970 to1972 and measurements made by the Bureau of Mines between 1967 and 1968. This was then combined with miner-reported work histories in order to create cumulative exposure estimates. A linear regression model that included cumulative dust exposure, smoking, age, and height as variables accounted for 47% of the total variability in observed FEV_1 values. From this model, the average decrement in FEV_1 was 0.69 mL per gh/m^3 of coal dust exposure (Attfield et al., 1992b).

Longitudinal studies have also been conducted using data from the PFR and NSCWP surveys. Love et al. (1982) examined lung function data from the first and third PFR surveys for five collieries. The first survey was attended by 6191 individuals, but only 2025 attended the second and third surveys. After exclusion of those with progressive massive fibrosis or inconsistent smoking histories, the remaining study sample consisted of 1677 men. Each man's cumulative dust exposure during and before the period of study was calculated using work histories and sampling data obtained between the first and third surveys. After adjusting for age, height, and smoking, rate of loss of FEV_1 over the 11-year observation period increased significantly with increasing previous dust exposure. It was estimated that exposure to the average level of dust of 117 gh/m^3 was associated with a 40 mL loss of FEV_1 over an 11-year period. For an average career exposure of 245 gh/m^3 (35 years work at a mean concentration of 4 mg/m^3), the estimated loss of FEV_1 in the subsequent 11 years was 87 mL (Love et al., 1982).

In the United States, Attfield (1985) conducted a longitudinal analysis of workers who participated in both the first and third NSCWP surveys. Of 9078 miners who took part in R1 (1969 to 1971), only 1470 also participated in the R3 survey (1977 to 1981). After limiting the analysis to only those workers aged 20-49 with dust exposure data from personal monitoring, 957 individuals made up the final study group, one-fifth of whom had radiographic pneumoconiosis. There were no significant differences with regards to demographic factors, pulmonary function, or respiratory symptoms between those miners who participated in R3 and those miners who took part in R1 but not R3. Because of the potential unreliability of the dust samples used in the NSCWP, several exposure indices were used: years underground before the initial survey, years worked at the coalface before the first survey, years worked at the coalface and underground between the two surveys, and average dust concentration derived from sampling between the surveys. The mean dust exposure for the 957 miners in the study group was 1.2 mg/m^3.

Depending on the exposure index used in the regression model, the estimated average excess reduction in FEV_1 in the 11-year period between surveys attributable to coal mining ranged from a 36 mL (the decline associated with 11 years of work underground) to 84 mL (the decline in the 11 years subsequent to working underground for 35 years). The author noted that previous studies had suggested that the general dust level in U.S. mines prior to 1969 was around 4 mg/m^3, and therefore this latter value of 84 mL closely corresponded to Love et al.'s (1982) estimate of 87 mL (Attfield, 1985).

These cross sectional and longitudinal studies may have underestimated the effects of coal dust exposure on FEV_1 decline due to the potential bias of only studying workers healthy enough to remain in the industry and participate in the surveys. This was especially noticeable in the longitudinal studies discussed above, since only 27% (Love et al., 1982) and 16% (Attfield, 1985) of miners who took part in initial surveys also participated in the final survey of the study. In order to address this possible survivor bias, Soutar et al. (1986) studied both active miners and ex-miners miners who took part in follow-up PFR surveys from 1974-1980; all subjects had previously participated in the third PFR survey (1963-1968). Just under two thirds of men who took part in the third survey participated in the follow-up survey. After exclusion of men with progressive massive fibrosis or unreliable information, 4059 men made up the study group: 1867 men (46%) were still working as miners, 1023 men (25%) were ex-miners less than age 65, and 1169 men (29%) were ex-miners greater than 65. When all men were considered, there was an inverse relationship between FEV_1 and dust exposure in all age and smoking groups. After adjusting for age, height, weight, and smoking, the estimated loss of FEV_1 per gh/m^3 was 0.76 mL, which was similar to the previously discussed cross sectional study estimates (Attfield et al., 1992b; Rogan et al., 1973). A recent review concluded that the study by Soutar et al. (1986) was probably the most reliable of the cross sectional analyses, due to the strength of the coal dust exposure data and the inclusion of ex-miners as subjects (Coggon et al., 1998).

2.1.2. Greater Dose-Response Estimates and Age Effects

These studies were fairly consistent in their estimates of the relationship between coal dust exposure and pulmonary function decline. For the miners exposed during their entire career to moderate coal dust concentrations of 4 mg/m^3, a modest average decline in FEV_1 of about 200 mL could be expected. However, coal dust effects of greater magnitude have been observed in more recent studies, despite more stringent coal dust exposure controls.

Seixas et al. (1992) conducted a cross-sectional study to determine the effects of lower levels of coal dust exposure experienced by coal miners who began working after the Federal Coal Mine and Safety Act took effect in 1969, which legislated that respirable coal dust exposures be less than 3 mg/m^3 starting in 1970, and less than 2 mg/m^3 by 1973. Of the 7387 miners who participated in either R1 or R2 in the 1970's, 1185 miners who had not worked in coal mines prior to 1970 and who took part in the R4 survey (1985 to 1988) were included in the analysis. Coal dust exposures were determined from personal samples obtained for compliance purposes from 1970 to 1987 and cumulative exposure was expressed as mg/m^3 – years. Corrections were made to account for previously identified potential compliance sampling biases (Seixas et al., 1990). The average cumulative coal dust exposure was 15.6 mg/m^3 – years. In a regression analysis adjusted for age, height, smoking status, years of smoking, race, and mining status, the estimated decline in FEV_1 was 5.5 mL per mg/m^3 – year of cumulative coal dust exposure. A log transformation of the exposure variable improved the model fit; there was a significant association between log cumulative exposure and FVC, FEV_1, and FEV_1/FVC, indicating that decrements in pulmonary function were proportionately greater in response to low cumulative exposures than to higher exposures (Seixas et al., 1992).

For comparison with the previously discussed studies, 1 mg/m^3 – years is equivalent to 0.575 gh/m^3. Using Soutar et al. (1986) as a reasonable benchmark exposure-response estimate from the previously discussed studies, their estimate of a 0.76 mL loss of FEV_1 per gh/m^3 is equivalent to a 1.3 mL loss of FEV_1 per mg/m^3 – year. The Seixas et al. (1992) exposure-response estimate was therefore about four times greater than earlier estimates. Seixas et al., (1992) suggested that their greater exposure-response estimate might have been greater due to a combination of factors, such as underestimation of exposure levels in U.S. coalmines. As well, their study population was younger and had worked for a shorter period of time than the Soutar et al., (1986) miners. If exposure early in a miners' career or at a young age were associated with a steeper decline in pulmonary function in response to coal dust exposure, then this also could have explained the higher exposure-response estimate (Seixas et al., 1992).

A follow-up study that included a longitudinal and cross sectional analysis examined a subset of these miners, consisting of 977 men who had performed pulmonary function tests at both R2 (1972-1975) and R4 (1985-1988) (Seixas et al., 1993). The average pulmonary function declines for this cohort during the period R2 to R4 were quite modest at 39 mL per year for FVC and 37 mL per year for FEV_1. In the longitudinal analysis, there were no significant associations between coal dust exposures experienced after R2 and pulmonary function changes. However, a cross sectional analysis of data from R4 confirmed the findings of the previous study (Seixas et al., 1992), as both FEV_1 and FEV_1/FVC were associated with cumulative dust exposure, with an estimated drop in FEV_1 of 5.9 mL per mg/m^3 – years.

Given the discrepancy between the findings from the longitudinal and cross sectional analyses, a further cross sectional analysis was performed using pulmonary function data from R2 and cumulative dust exposure from before R2. Even though the average exposure time was 2.5 years (maximum exposure time 5 years), there was a strong association between dust exposure and declines in FEV_1

and FVC, with each being about 30 mL lower for each additional mg/m^3 – year of exposure. When this group was stratified into those workers < 25 years of age at R2 and those > 25, marked differences were seen: FEV$_1$ and FVC declines for those < 25 were around 15 mL per mg/m^3 – years and not significant whereas for those > 25, FEV$_1$ and FVC declines were around 40 mL per mg/m^3 – years and significant. From this study, it was concluded that miners experienced a rapid initial loss of lung function in the first few years of exposure to coal mine dust, with continued exposures having little additional effect on lung function decline (Seixas et al., 1993). This was also alluded to in the previous study by the same authors: an interaction with log cumulative exposure and age and smoking was found, suggesting that dust-related declines in pulmonary function were greater in young miners and non-smokers (Seixas et al., 1992).

Building on these findings, Henneberger et al. (1996) attempted to determine if new miners were more susceptible to the effects of coal dust than experienced miners. Similarly to Seixas et al. (1993), miners who participated in R4 and also in R1 or R2 were considered eligible for study. However, the miners who were hired after 1970 and previously examined (Seixas et al., 1992; Seixas et al., 1993) were excluded. Of the 2778 miners who started mining before 1970, 1915 subjects made up the study group. Although all subjects were mining at R1 or R2, only half were still mining at R4. The mean cumulative dust exposure at R1/R2 was 38.5 mg/m^3 – years. The mean exposure between R1/R2 and R4 was 0.9 mg/m^3.

A cross sectional analysis of the R1/R2 data revealed a trend toward declining FEV$_1$ and increasing FVC with each unit of dust exposure, but this was not significant. Using only R4 data, FEV$_1$ decline was significantly associated with cumulative dust exposure, with an estimated loss of 1.2 mL per mg/m^3 – year. There was an interaction between dust exposure and smoking and FEV$_1$/FVC, such that the decline in FEV$_1$/FVC (-0.038% per mg/m^3 – year) was limited to current smokers. In a longitudinal analysis, statistically significant declines in FEV$_1$ and FVC (0.07 and 0.10 mL per mg/m^3 – year, respectively) were associated with pre-R1/R2 cumulative dust exposure, but not post-R1/R2 exposure (Henneberger et al., 1996).

When the miners in this study were compared with miners hired after 1970 and studied by Seixas et al. (1993), numerous differences were apparent. The miners hired before 1970 had a pre-R1/R2 mean cumulative coal dust exposure of 38.5 mg/m^3 – years, whereas the corresponding figure for the newer miners was 3.8 mg/m^3 – years. Even though both groups of miners had the same post R1/R2 exposures, the large discrepancy in the pre-R1/R2 exposures meant that by R4, the older miners had a mean cumulative dust exposure that was over three times greater than those miners hired after 1970 (52.2 mg/m^3 – years versus 15.4 mg/m^3 – years). However, despite the lower exposures experienced by the newer miners, the estimated rate of FEV$_1$ decline for new miners was 5.9 mL per mg/m^3 – year, almost five times greater than the estimate for older miners of 1.2 mL per mg/m^3

– year. In the Discussion section of their paper, Henneberger et al. (1996) suggest that the results obtained from the older miners may have been biased by the healthy worker effect: if miners who had developed work-related respiratory symptoms had left the mining industry, the estimates of pulmonary function decline would have been based only on healthier workers, thereby potentially underestimating the effect of cumulative coal dust exposure. It has also been postulated that the differences in pulmonary function decline between older and newer miners may be due to errors in exposure measurements, or alternatively, that a true biologic phenomenon exists (Cohen et al., 2002).

The same subjects were included in a follow-up paper that examined the association of respiratory symptoms with coal dust exposure (Henneberger et al., 1997). Unlike the previous study that used continuous exposure measures, dust exposures in this study were categorized based on both pre-R1/R2 and post-R1/R2 exposures. Pre-R1/R2 exposure categories were 0-12.7, 12.8-33.4, and 33.5-197.7 mg/m^3 – year, and post-R1/R2 categories were 0-12.7 and 12.8-33.4 mg/m^3 – year, resulting in 6 cells of pre- and post-R1/R2 exposure combinations. Higher pre-R1/R2 exposures were associated with greater declines in FEV_1 (-100 mL for level III and –51 mL for level II as compared to the lowest exposure level). In contrast, the difference in FEV_1 decline for post-R1/R2 categories was only 8 mL, suggesting that early exposures have a greater negative impact on FEV_1 than more recent exposures. With regards to respiratory symptoms (chronic bronchitis, shortness of breath, wheeze), however, both early and later exposures demonstrated adverse effects. The prevalence of respiratory symptoms increased with higher levels of both pre- and post-R1/R2 exposures. There was a statistically significant increased risk for each symptom when comparing the highest and lowest category for both pre and post R1/R2 exposures (Henneberger et al., 1997).

The adverse effects of relatively low coal dust exposures, especially during the initial years of mining work, were recently demonstrated in a study of Italian miners (Carta et al., 1996). This was a longitudinal study of 909 miners who had worked for at least two years in a Sardinian coal mine that opened in 1977 and who participated in at least three of seven surveys conducted between 1983 and 1993. Chest radiographs and pulmonary function tests were performed and data on respiratory symptoms, occupational histories, and smoking were collected at each survey. None of the miners had radiographic pneumoconiosis. The mean duration of follow-up was 7 years, with 40% of subjects providing 10 years of observation. Respirable dust concentrations at the coal face ranged between 1.73 to 3.05 mg/m^3, whereas for other underground workers and surface jobs, the average exposures were less than 1.0 mg/m^3. This was one of the few studies to measure exposures other than coal dust: respirable quartz concentrations were greater than 0.1 mg/m^3 in one quarter of coal face samples and less than 12% of other underground samples, and the vast majority of samples for nitrogen oxides, sulphur dioxide, aldehydes, and polycyclic aromatic hydrocarbons were below their respective time-weighted average thresholds.

After controlling for initial lung function values, age, and smoking, individual exposure to respirable coal dust had a significant effect on the decline of FVC and FEV_1. The average annual decline in lung function over the period of study was 5.7 mL for FVC and 7.6 mL for FEV_1 per mg/m^3 of coal dust exposure (Carta et al., 1996). However, the rate of lung function decline was inversely related to initial cumulative exposure to dust, suggesting that dust exposures during the longitudinal follow-up period had a greater effect on lung function decline in miners with no or very little previous dust exposure, as compared to miners with higher initial cumulative exposures. The authors postulated that this finding could be consistent with a greater decline in lung function during the initial years of coal dust exposure, which has been supported by previous studies (Henneberger et al., 1996; Henneberger et al., 1997; Seixas et al., 1993).

Similar conclusions were drawn from a recent cross sectional study that compared 1286 British coal miners over age 40 who underwent pulmonary function studies and chest x-rays in 1992 to 1993 with a local comparison group of men aged 40-70 who had never worked in mining or other dusty trades. None of the miners had radiographic pneumoconiosis (Lewis et al., 1996). In an analysis that adjusted for age, height, and smoking, the overall effect of mining was a decrement in FEV_1 of 155 mL. When only non-smokers were considered, the mean effect of mining exposure was a 128 mL decrement in FEV_1. There was an interaction between age and mining exposure, such that the effect of mining was greater in younger miners; when only subjects under age 45 were considered, the independent effect of mining was a decline in FEV_1 of 251 mL (Lewis et al., 1996).

2.1.3 Survival Bias and Clinical Significance

Henneberger et al. (1996) had suggested that the greater decline in pulmonary function observed in newer miners as compared to older miners was due to the healthy worker effect: miners that develop respiratory symptoms and leave the mining industry would not be included in studies of older miners, potentially underestimating the effect of cumulative coal dust exposure. Others have also suggested the possibility of selection, or survival, bias in studies of coal miners (Attfield et al., 1996b; Oxman et al., 1993; Stenton et al., 1993). The larger studies of British and U.S. coal miners concluded that the average ventilatory impairment attributable to a working career of coal dust exposure was only in the range of about 200 mL in FEV_1 and not clinically significant (Attfield et al., 1992b; Rogan et al., 1973). If a survivor bias were present, however, then the true clinical significance of ventilatory function loss in susceptible subpopulations of miners would be underestimated. As well, Stenton et al. (1993) point out that in the case of smoking, only a small proportion of individuals suffer adverse pulmonary function effects, whereas the majority are not affected at all. If the same were true of the effects of coal dust exposure, then the observed modest average decline in FEV_1 could be the result of deficits experienced by a minority of severely affected workers, with most not affected at all.

In the previously mentioned study by Soutar et al. (1986) that examined miners and ex-miners, the estimated loss of FEV_1 per gh/m^3 was 0.76 mL after adjusting for age, height, weight, and smoking. This estimate was equivalent to a loss of 228 mL of FEV_1 for a moderately high lifetime exposure of 300 gh/m^3. Ex-miners with chronic bronchitis, however, had more marked pulmonary function decrements. Estimated losses of FEV_1 for a lifetime dust exposure of 300 gh/m^3 in this select subgroup were 492 mL for smokers, 942 mL for ex-smokers, and 420 mL for non-smokers, equivalent to FEV_1 declines per gh/m^3 of 1.64 mL, 3.14 mL, and 1.40 mL, respectively. Although the small group of symptomatic ex-miners were not typical of miners generally, the authors felt that the effects seen were representative of the effects of dust exposure in susceptible men (Soutar et al., 1986).

Susceptible miners were examined in greater detail in a study focussed specifically on the 199 ex-miners with chronic bronchitis from the Soutar et al. (1986) study who had left coal mining prior to age 65 and found other work (Hurley et al., 1986). These ex-miners did not have unusually high dust exposures as compared to the larger cohort of 4059 men, but their FEV_1 was lower than those miners who remained in the industry. In a multiple regression analysis, the influence of dust exposure on FEV_1 in this select group of ex-miners was more highly statistically significant than the effects of age, height, weight, and smoking. The dust exposure estimate was a 2.0 mL decline in FEV_1 per gh/m^3. This was equivalent to a 316 mL loss of FEV_1 for the average dust exposure of the group or a 600 mL decrement for a moderately high exposure of 300 gh/m^3. These estimates were based on dust exposure alone, and greater decrements would be expected if the effects of age and smoking were also included. The authors stressed that although respiratory impairment from coal dust exposure was severe in this select group of workers, the average response of FEV_1 to dust exposure was clinically modest (Hurley et al., 1986).

In the United States, attempts have also been made to identify miners who are more susceptible to the adverse effects of coal dust on pulmonary function. Potential risk factors for increased susceptibility were explored in a nested case-control study of NSCWP miners (Wang et al., 1999a). Using a 6 to 18 year follow-up period that ended between 1977 and 1988, cases were those miners whose annual rate of decline in FEV_1 differed by more than 60 mL/year from referent miners who were matched for survey, age, height, smoking status, and initial FEV_1. The original cohort consisted of 634 eligible miners, but only 264 miners (42%) completed a questionnaire survey conducted between 1994 and 1997 that explored work practices and medical history. As compared to referents, cases had gained 6 pounds more weight by the final survey, were less likely to wear a respirator, had greater exposure to face work, shotfiring, roof bolting, and hand loading, and worked for a longer period of time in mines that used water from holding tanks for dust control. Cases were also more likely to have had pneumonia before age 16 and to have been exposed to environmental tobacco smoke and indoor fuel smoke as children. Significant determinants that were independently associated with case status were non-use of a respirator, duration of underground mining, exposure to roof bolting, and childhood domestic exposure to tobacco smoke (Wang et al., 1999a).

The morbidity and mortality experience of the same subjects were assessed in a follow-up study (Beeckman et al., 2001). A higher proportion of cases had left mining before retirement age due to respiratory illness (17.1% vs. 5.6% for referents). For cases, the cumulative incidence of respiratory symptoms (cough, phlegm production, dyspnea, and wheezing) and self-reported diagnoses (bronchitis, asthma, emphysema) were each approximately double that of controls. Cases were also more likely to die of cardiovascular disease and non-malignant respiratory disease. After adjustment for age and smoking, a coal miner with a rapid decline in FEV_1 over an average observation period of 11 years would have twice the risk of dying of cardiovascular disease or non-malignant respiratory disease, and a 3.2 times greater risk of dying of chronic obstructive pulmonary disease than a miner with a more stable FEV_1 over the same follow up period (Beeckman et al., 2001).

Attempts have been made to gauge the clinical significance of observed ventilatory deficits by comparisons with predicted lung function values. For example, Seixas et al. (1992) used linear regression analyses with categorical outcomes, such as FEV_1 <80% predicted and FEV_1/FVC <80% predicted (using prediction equations of Crapo et al., 1981). For an increase in cumulative exposure of 20 mg/m^3 – years, elevated odds ratios in the 1 to 2 range were obtained for all pulmonary function and respiratory symptom outcome measures, and for $FEV_1/FVC < 80\%$, the odds ratio was 2.5. Separate analyses were performed with the cohort stratified into current, ex, and never smokers. Significant associations between cumulative exposure and pulmonary function decline or pulmonary symptoms were found for never smokers, but not current smokers. It was suggested this might have been explained by the effects of smoking masking the more variable effect of dust exposure (Seixas et al., 1992).

From the longitudinal analysis of Italian miners, Carta et al. (1996) reported that for men who were asymptomatic at their first survey, the odds of developing respiratory symptoms (shortness of breath while walking on level ground, chronic bronchitis, or wheeze) were greater with increasing cumulative dust exposure. When exposure was divided into quartiles, age and smoking adjusted odds ratios for symptom onset were around 1.8, 2.0, and 2.5 for 0.74, 0.93, and 1.13 mg/m^3 – years of exposure to respirable coal dust, respectively (Carta et al., 1996).

The same group of workers initially examined by Rogan et al. (1973) was analyzed further using a subgroup consisting of 451 lifetime non-smokers without bronchitis to generate internally-derived predicted FEV_1 values based on age and height (Marine et al., 1988). The proportion of workers with respiratory dysfunction indices, such as FEV_1 <80% and FEV_1 <65% predicted, increased with increasing levels of cumulative dust exposure. Smokers had a higher prevalence of respiratory dysfunction for a given dust exposure, but the exposure-response trend was similar for both smokers and non-smokers. The prevalence of workers with FEV_1 <65% was 3.2% and 7.7% for non-smokers with zero and high exposures, respectively, and 5.0% and 14.2% for smokers with zero and high exposures, respectively (Marine et al., 1988).

Based on these results, it was estimated that 8% and 6.6% of non-smokers and smokers, respectively would have an FEV_1 <80% predicted after a cumulative coal dust exposure of 122.5 gh/m^3 (equivalent to 2 mg/m^3 coal dust exposure over a 35 year career) (Oxman et al. 1993). The corresponding percentages of non-smokers and smokers who would end up with an FEV_1 <65% predicted were 1.2% and 2.3%, respectively (Oxman et al., 1993).

Internally derived predicted FEV_1 values were also used in a later study of British miners (Soutar et al., 1993). From 1981 to 1986, 1671 miners at 3 PFR collieries where dust measurements continued after 1980 underwent medical examinations. Predicted FEV_1 values were calculated according to age, height, and weight, assuming non-smoker miner status without respiratory symptoms. The difference between the predicted FEV_1 value and the average age, height, and weight-adjusted FEV_1 of miners with exertional dyspnea was used as the cut-off of a clinically significant FEV_1 deficit. The proportion of subjects with FEV_1 deficits that were at least as severe as the deficit associated with severe exertional dyspnea ranged from 12 to 24% at the three collieries. Such cases were more frequent among ex-miners than current miners and among smokers than non-smokers. The risk of having a severe FEV_1 deficit increased with increasing cumulative dust exposure.

In a cross sectional study of 7188 miners from the fifth PFR survey (1973 to 1977), the association between FEV_1 and cumulative dust exposure was adjusted for age, physique, and smoking using multiple linear regression (Soutar et al., 2004). This study defined clinically important deficits in FEV_1 using comparisons with reported symptoms of breathlessness. A threefold increase in the odds of reporting "walking slower than other people on level ground because of their chest" was associated on average with a 993 mL FEV_1 deficit from predicted. Risk of at least this decrement in FEV_1 by age 60 for non-smokers was 10% for no dust exposure, increasing to 19% after a working career exposed to an average dust concentration of 6 mg/m^3. The corresponding numbers for smokers were 22% and 36%, respectively. A twofold increase in risk of reporting breathlessness was associated with an average loss of FEV_1 of 627 mL. The risk of experiencing this decrement in FEV_1 after a working career at 6 mg/m^3 was 40% for non-smokers and 60% for smokers (Soutar et al., 2004).

Lewis et al. (1996) used predicted pulmonary function values based on age and height that were calculated from the multiple linear regression model of miners and control subjects who were 45 years of age or less. Based on this prediction equation, significantly more miners than controls (4.7% vs. 0.7%) had an observed FEV_1 value that was one litre or more below the predicted value (Lewis et al., 1996).

Taken together, these studies suggest that a small proportion of miners may be more susceptible to the adverse effects of coal dust exposure on pulmonary function, leading to clinically significant impairment, respiratory symptoms, and greater risk for mortality due to cardiovascular and pulmonary disease.

2.1.4. Influence of Radiographic Pneumoconiosis

It has been demonstrated consistently that the presence of small opacities on chest radiography, consistent with simple Coal Workers' Pneumoconiosis, does not greatly influence the relationship between coal dust exposure and lung function decline.

In the study of 3581 coal miners who took part in the third PFR survey (Rogan et al., 1973), it was observed that men with radiological pneumoconiosis had lower FEV_1 levels than those without, but this was attributed to the fact that men with pneumoconiosis were older and had greater cumulative dust exposures. There was no evidence that those with pneumoconiosis experienced any loss of lung function in excess of that attributable to their dust exposures (Rogan et al., 1973).

As previously discussed, in Soutar et al.'s (1986) study of both active and ex-miners, the estimated loss of FEV_1 per gh/m^3 of coal dust exposure was 0.76 mL after adjusting for age, height, weight, and smoking. This estimate increased to 1.06 mL loss of FEV_1 per gh/m^3 in a subset of 2877 men without radiological evidence of pneumoconiosis. A similar observation was made by Attfield et al. (1992b) in their study of U.S. coal miners. When a subset of 4913 miners without radiographic evidence of pneumoconiosis were examined separately, the estimated FEV_1 decline was 0.75 mL per gh/m^3, a value which was very similar to that obtained for the overall cohort, 0.69 mL per gh/m^3.

Studies of British (Soutar et al., 1986, Lewis et al., 1996), U.S. (Attfield et al., 1992b), Italian (Carta et al., 1996), and Belgian (Nemery et al., 1987) coal miners have all demonstrated reductions in pulmonary function attributable to coal dust exposure in the absence of radiological pneumoconiosis. Recent reviews have concluded that studies to date are consistent with hypothesis that although pneumoconiosis is a good marker for exposure to dust, the respiratory effects of dust on spirometric indices are the same in dust-exposed workers regardless of the presence of radiographic pneumoconiosis (Cohen et al., 2002; Oxman et el., 1993).

2.1.5 Influence of Smoking

The influence of smoking status on the relationship between coal dust exposure and pulmonary function decrements has been examined in the vast majority of studies on coal miners, either through regression analyses or subject stratification. All of the previously discussed estimates of the effect of cumulative coal dust exposure on pulmonary function were derived from studies that controlled for smoking in their regression models (Attfield, 1985; Attfield et al., 1992b; Carta et al., 1996; Henneberger et al., 1996; Love et al., 1982; Rogan et al., 1973; Seixas et al., 1992; Soutar et al., 1986).

In some studies, the observed effects of coal dust exposure have been greater in non-smokers as compared to current smokers (Attfield et al., 1992b; Seixas et al., 1992). For example, when never smokers, ex-smokers, and current smokers were analysed in separate regressions, the effect of dust exposure on FEV_1 was more severe in never and ex-smokers (-0.73 mL and -1.0 mL per gh/m^3, respectively) than current smokers (-0.44 mL per gh/m^3) (Attfield et al., 1992b). It has been suggested that the effects of smoking might mask the effects of dust exposure (Seixas et al., 1992).

It has also been suggested that the effects of coal dust exposure in non-smokers closely approximates the respiratory function decline due to smoking. For example, in a longitudinal study of U.S. miners, the decline in FEV_1 over an 11-year period was estimated to be 84 mL, whereas the equivalent effect of smoking was an excess FEV_1 decline of about 100 mL (Attfield, 1985). Studies of both U.S. and British coal miners have concluded that working for one year in an underground coal mine can result in an FEV_1 decrement that is equivalent to one pack-year of smoking (Attfield et al., 1992b; Lewis et al., 1996). Marine et al. (1988) observed that for non-smokers with high dust exposure, the prevalence of respiratory dysfunction (eg. FEV_1 <80% predicted, FEV_1 <65% predicted) was equivalent to that of smokers with no dust exposure. Overall, the effects of dust exposure was similar for smokers and non-smokers, and the effects of smoking and dust exposure appeared to be additive (Marine et al., 1988).

Smaller studies from France and Belgium have also noted that coal dust effects can be observed in the absence of smoking and that smoking adds to the pulmonary function decrement. A longitudinal study was performed on a select group of coal miners in Lorraine, France who were only referred to the pulmonary function laboratory because of either radiographic changes or a symptomatic complaint of breathlessness (Bates et al., 1985; Dimich-Ward et al., 1994). Assessments were performed between 1950 and 1982 on 397 miners with at least 20 years of mining experience. In this select group of miners, annual declines in FEV_1 were elevated, ranging from – 46 mL/year in non-smokers to –58 mL/year in smokers (Bates et al., 1985). A re-analysis of the data that controlled for smoking, age, height, length of follow-up, and time since retirement demonstrated that the elevated estimates of FEV_1 decline were only slightly more severe (an additional 5 mL/year) for smokers as compared to non-smokers (Dimich-Ward et al., 1994). After retirement and removal from further coalmine exposure, the rate of pulmonary function decline was 50 and 56 mL/year in non-smokers and smokers, respectively.

A smaller study of lifetime non-smokers compared 32 Belgian coal miners with 32 steelworkers, with age, height, and weight similar between the two groups (Nemery et al., 1987). FEV_1, FEV_1/FVC, and measures of maximal expiratory flow rates were all significantly lower in the coal miners as compared to the steelworkers. For example, the average coal miner FEV_1 was 3.89 L, as compared to the average steelworker FEV_1 of 4.32 L.

2.1.6. Coal Summary

The majority of studies that have explored the relationship between coal dust exposure and pulmonary function have been derived from a series of cross-sectional surveys conducted in the United Kingdom (Pneumoconiosis Field Research) and the United States (National Study of Coal Workers' Pneumoconiosis). Several of these have estimated that average decrements in FEV_1 attributable to coal dust exposure are relatively modest at around 0.6 to 1.0 mL per gh/m^3 (1.0 to 1.7 mL per mg/m^3-year) of coal dust exposure, approximately equivalent to a 200 mL decline in FEV_1 over a working career (Attfield et al., 1992b; Marine et al., 1988; Rogan et al., 1973; Soutar et al., 1986). Although these studies may have been influenced by survivor bias since they only surveyed active miners, a study that included ex-miners as well observed a similar exposure-response relationship (Soutar et al., 1986).

However, several studies published within the past fifteen years have indicated that the effect of coal dust exposure on pulmonary function may not be uniform over a working career. Dust-related declines in pulmonary function appear to be greatest in younger miners and during the initial years of exposure (Carta et al., 1996; Henneberger et al., 1996; Henneberger et al., 1997; Seixas et al., 1992; Seixas et al., 1993). There is also evidence that although declines in pulmonary function due to coal dust exposure may be modest when averaged over a cohort of miners, a small subset of individuals may be more susceptible to dust effects and develop clinically significant respiratory impairment (Beeckman et al., 2001; Hurley et al., 1986; Soutar et al., 1986; Soutar et al., 1993; Soutar et al., 2004; Wang et al., 1999a).

Increasing cumulative coal dust exposure is associated with an increased risk of developing Coal Workers' Pneumoconiosis, but this appears to be independent of the effects of coal dust exposure on pulmonary function. Decrements in pulmonary function can occur in the absence of radiographic pneumoconiosis, and the presence of CWP does not cause an incremental loss of lung function over and above what is attributable to dust exposure (Cohen et al., 2002; Oxman et al., 1993; Rogan et al., 1973).

It is estimated that declines in pulmonary function due to work at the coal face in underground mining is equivalent in magnitude to the effects of smoking. Both non-smokers and smokers can experience adverse pulmonary effects from coal dust exposure, but miners who smoke suffer greater decrements in FEV_1 than non-smoking miners; the effects of the coal dust exposure and smoking appear to be additive (Attfield et al., 1992b; Love et al., 1982; Marine et al., 1988; Seixas et al., 1992; Soutar et al., 1986).

2.2 Silica

Silica refers to the chemical compound silicon dioxide, the earth's most abundant mineral, which may exist in a crystalline or noncrystalline (amorphous) form. Several forms of crystalline silica exist, including alpha quartz, beta quartz, cristobalite, tridymite, ceosite, moganite, and stishovite. Alpha quartz is the most abundant form found in nature and is a component of virtually every mineral deposit (NIOSH, 2002). Any process that involves movement of earth or disturbance of silica-containing products may expose a worker to silica; the most common occupations at risk of silica exposure include mining, milling, quarrying and stone work, tunneling, foundry work, sandblasting, pottery making, glass making, and boiler work (Banks et al., 2005).

Inhaled crystalline silica particles that are small enough to reach the level of the respiratory bronchioles can initiate an inflammatory reaction. Key factors that influence the biological response to silica in addition to particle size are the dose and duration of exposure, crystalline structure, presence of highly reactive surface groups, and persistence in lung tissue. Silica particles can be directly cytotoxic, reacting with cell membranes to cause cell injury, oxidant damage, and release of inflammatory mediators. The most common health effect associated with long-term inhalation of dust containing crystalline silica is silicosis, a diffuse interstitial fibronodular lung disease (Banks et al., 2005). The primary event in the disease process is the interaction of the silica particle with the lung macrophage. After silica particles are phagocytized, the macrophage becomes activated, causing oxidant damage and releasing mediators that initiate a cascade of inflammatory events including leukocyte recruitment and fibrotic responses (NIOSH, 2002). Dust-laden macrophages eventually collect in an arrangement that is surrounded by a reticulum of fibrous tissue, known as a silicotic nodule. Active inflammation continues at the periphery of the lesion, and as nodules enlarge and coalesce, they may form conglomerate lesions that may encroach on airways and pulmonary vasculature, leading to pulmonary impairment. The presence of conglomerate lesions greater than 1 centimeter is indicative of complicated silicosis, also known as Progressive Massive Fibrosis (PMF) (Banks et al., 2005).

Typically, silicosis does not appear until a threshold exposure level has been reached. Histologic observations suggest that the inflammatory disease process described above is not evident in people with low levels of exposure to silica dust, presumably because the normal pulmonary defense mechanisms are able to remove inhaled dusts until the exposure level becomes relatively high, thereby overwhelming clearance mechanisms (Mossman et al., 1998).

Silicosis is broadly classified into three main types. The most common is chronic silicosis, characterized by discrete nodular lesions, typically 4 to 10 millimeters in diameter, and more predominant in the upper lobes of the lungs. These lesions are usually manifest after 10 to 30 years of chronic exposure to crystalline silica. These nodular lesions can be visualized on a chest radiograph, and their profusion is the basis for the ILO classification of silicosis. The remaining forms of silicosis, accelerated and acute, are rare but more severe. Acute silicosis occurs after exposure to high concentrations of free silica dust and presents with rapidly progressive dyspnea and respiratory insufficiency secondary to lipid-rich pulmonary edema and interstitial inflammation. Accelerated silicosis develops within 2 to 5 years of intense silica exposure and PMF is more commonly observed than chronic silicosis. Both acute and accelerated silicosis are invariably fatal (Davis, 1996; Ding et al., 2002).

Although the most common health effect of occupational exposure to crystalline silica is silicosis, other adverse health effects associated with crystalline silica exposure include pulmonary tuberculosis (silico-tuberculosis), bronchitis, bronchogenic carcinoma, autoimmune diseases such as systemic sclerosis, rheumatoid arthritis, and systemic lupus erythematosus, and renal disease (ATS, 1997; Banks et al., 2005; Ding et al., 2002; Steenland , 2005).

2.2.1 Radiographic Silicosis and Exposure-Response

Silicosis was first reported by the ancient Greeks and has been recognized throughout history (Banks et al., 2005). However, it has been noted that despite the abundance of historical literature on silicosis, very little work has been done to explore the quantitative relationship between exposure and disease (Steenland et al., 1995). Several recent studies have tried to elucidate the exposure-response relationship between cumulative silica exposure and radiographic silicosis. In Canada, Muir and colleagues examined 2109 Ontario hard rock miners (predominantly from gold and uranium mines) (Muir et al., 1989a; Muir et al., 1989b; Verma et al., 1989). The miners included in the cohort started work after 1940 and before 1960, and they were followed up until the end of 1982. Cumulative dust exposure for each miner was determined based on work history and dust measurements and expressed as mg/m^3-years. Silicosis was defined as a chest radiograph with an ILO profusion category of 1/1 or greater. Chest x-rays were obtained annually from actively employed Ontario miners since 1927. Out of 2109 miners, silicosis was identified in 32 (1.5%). Cumulative respirable silica exposure estimates that would result in a 1%, 2%, 5%, and 10% risk of developing silicosis were 2.1, 3.3, 6.0, and 9.6 mg/m^3-years, respectively. Cumulative risk estimates for developing silicosis were 0.9%, 2.7%, 5.0%, and 7.7% after 40 years of exposure to 0.05, 0.10, 0.15, and 0.20 mg/m^3 respirable silica, respectively (Muir et al., 1989b).

These risk estimates have been considerably lower than subsequent analyses. A study of 2235 South African gold miners followed past retirement observed that 313 miners (14%) developed radiographic silicosis (ILO profusion category 1/1 or greater) (Hnizdo et al., 1993). Only 43% of cases were diagnosed with silicosis at an average age of 51, whereas in 57% of cases, the diagnosis was made an average of 7.4 years after leaving the mines at an average age of 59. In this cohort, the estimated cumulative risk of silicosis was 25% at a cumulative respirable silica exposure of 2.7 mg/m^3-years. This cumulative risk rose to 77% at 4.5 mg/m^3-years of cumulative respirable silica exposure (Hnizdo et al., 1993).

These results were closely mirrored by a subsequent study of 3330 South Dakota gold miners who had an average length of employment of 9 years and an average follow-up of 37 years (Steenland et al., 1995). Silicosis cases were identified from death certificates (with silicosis identified as an underlying or contributing cause of death) or through chest x-rays taken at two cross-sectional surveys conducted in 1960 and 1976. In this cohort, 170 (5%) silicosis cases were identified. The cumulative risk of silicosis rose steadily with cumulative exposure, with risk estimates of 1.7%, 6.0%, 6.0%, 40.3%, and 67.8% for cumulative respirable silica exposures of 0.5 to 1.0, 1.0 to 2.0, 2.0 to 3.0, 3.0 to 4.0, and more than 4.0 mg/m^3-years, respectively (Steenland et al., 1995).

It has been suggested that the study by Muir et al. (1989b) had lower risk estimates because the follow-up period was shorter and silicosis cases were only detected among active workers (Hnizdo et al., 1993; Steenland et al., 1995). This is supported by a recent review that noted that studies that did not include follow-up of workers after employment substantially underestimated silicosis risk (Steenland, 2005). Based on data from studies with adequate follow-up, the risk of developing silicosis after a 45-year exposure to 0.1mg/m^3 respirable silica (the current United States standard) ranged from 47% to 77% (Steenland, 2005). The inadequacy of current exposure standards for the prevention of silicosis was also highlighted by a recent pooled analysis of six cohorts, which included a total of 170 silicosis deaths out of a population of 18 364 workers. The estimated cumulative risk of death due to silicosis was 13 per 1000 following an exposure of 0.1mg/m^3 respirable silica from age 20 to 65 (equivalent to a cumulative respirable silica exposure of 4.5 mg/m^3-years) (Mannetje et al., 2002). Overall, existing data indicate that current occupational exposure limits for crystalline silica are not protective enough to prevent an unacceptable burden of silicosis among workers.

2.2.2 Pulmonary Function and Exposure Response

2.2.2.1 Miners

In addition to associations between silica exposure and radiographic silicosis, several studies have explored the relationship between exposure and pulmonary function. These studies are heterogeneous with respect to data quality and industry group studied. One group of workers that has received considerable research attention are South African gold miners, because these workers were exposed to dust with a high concentration of crystalline silica; silica made up 60% of the virgin rock and 30% of the respirable dust (Hnizdo et al., 1990). Four of these studies analyzed white miners who presented for medical examinations at the Medical Bureau for Occupational Diseases between 1968-1971 and who satisfied the following criteria: aged 45-54, worked underground for at least 10 years, lived in South Africa for at least 20 years, worked for less than 2 years in mines other than gold, and entered into the mining industry between 1936 and 1943 (Hnizdo et al., 1990; Hnizdo, 1992; Irwig et al., 1978; Wiles et al., 1977). Annual medical examinations were compulsory for all miners in dusty occupations, and miners who were retired and seeking compensation were also included in the selection process. Data collected during these surveys included occupational and smoking histories, respiratory symptoms, respiratory function measurements, and chest radiographs. Dust exposures were determined by dividing the miners into 11 occupational groups, and then measuring the personal dust exposure for a random sample of men from each group over an entire shift. It has been noted that there was a significant relationship between silicosis and these occupational groupings (Wiles et al., 1977). The average concentrations of respirable silica in the South African underground gold mining industry ranged from 0.05 to 0.84 mg/m^3 (Hnizdo et al., 1990).

Irwig et al. (1978) performed a cross-sectional study of 1973 men, 134 of whom had radiographic silicosis (ILO profusion category 1/0 or greater), in an attempt to determine if respiratory symptoms and lung function differed between men with silicosis and those without. On average, men with silicosis had a 41% higher cumulative exposure to dust than those without silicosis (dust concentrations were measured as respirable surface area, the total surface area in the respirable fraction of the dust, calculated from the observed projected area of particles, assuming them to be spherical). Mean FVC did not differ between the two groups, but those with silicosis had significantly lower FEV_1 and $FEF_{25\text{-}75\%}$ by 5% and 14%, respectively. Miners with silicosis were then matched to miners without silicosis, based on age, height, cumulative dust exposure, and smoking. In this matched analysis, there were no significant differences in FVC, FEV_1, and $FEF_{25\text{-}75\%}$ between miners with silicosis and those without. The authors concluded that the presence of pneumoconiosis was not associated with a greater decline in pulmonary function than what was directly attributable to dust exposure (Irwig et al., 1978).

Wiles et al. (1977) studied 2209 South African gold miners in order to determine if there was a dose-response relationship between obstructive lung disease and dust exposure (this study population was also used for the silicosis risk assessment of Hnizdo et al. (1993), discussed above). Similarly to Irwig et al., (1978), cumulative dust exposure was expressed as respirable surface area multiplied by the duration of exposure, to give a unit of RSA-years. The main measures of pulmonary function were FEV_1 and MMEF, and the absolute value of both declined significantly with increasing cumulative exposure. MMEF dropped steadily from 3.23 L/s at an exposure of 10000 RSA-years to 2.49 L/s at 38000 RSA-years. For FEV_1, the corresponding volumes for the equivalent cumulative exposure levels were 3.40 L and 2.97 L, respectively. At the highest cumulative exposure level, the declining trend for FEV_1 and MMEF, reversed slightly (3.04 L and 2.60 L/s, respectively, at 52000 RSA-years). The group was then stratified by smoking habit, and similar significant declines in MMEF with increasing cumulative dust exposure were observed (FEV_1 was not reported in this analysis) regardless of smoking status. At the lowest dust exposure level of 10000 RSA-years, the MMEF was 4.20 L/s, 3.22 L/s, and 2.96 L/s for non-smokers, ex-smokers, and smokers, respectively, dropping to 3.08 L/s, 2.75 L/s, and 2.32 L/s, respectively at a cumulative exposure of 38000 RSA-years (Wiles et al., 1977). Age was not controlled for in the analysis for this study.

The Wiles et al. (1977) dataset was reanalyzed by Hnizdo et al. (1990) using a reference group of 483 male nonminers drawn from municipal and government departments. Prediction equations were derived from 731 healthy miners by the Lung Function Unit of the Medical Bureau of Occupational Diseases and resulted in predicted lung function values that were very similar to those derived by Crapo et al. (1981). Declines in VC, FEV_1, FEV_1/VC, and $FEF_{25-75\%}$ occurred in a dose-response manner up to 40 000 RSA-years, and then increased slightly at higher exposure levels, “presumably due to some systematic effect, possible a healthy worker effect” (Hnizdo et al., 1990). Although the absolute values differed across smoking categories, the slopes were parallel. In a regression analysis that controlled for age, height, weight, and smoking, FEV_1, VC, and $FEF_{25-75\%}$ declined by 6.7 mL, 5.2 mL, and 11.8 L/s, respectively per 1000 RSA-year increase in cumulative exposure. There was no significant interaction between smoking and dust exposure. However, when miners were grouped according to lung function impairment category based on predicted lung function values, there appeared to be a synergistic effect between smoking and dust exposure. For example, the odds ratios for “marked obstruction” (normal VC, $FEV_1 < 99\%$ confidence limit, and $FEV_1/VC < 95\%$ confidence limit), for the two highest dust exposure and smoking categories (OR = 26 to 34) were approximately equal to the products of the marginal odds ratios (OR = 2.0 to 2.9 for high dust exposure categories, and OR = 8.4 to 10.4 for high smoking categories), indicating that the combined effects were multiplicative (Hnizdo et al., 1990).

A subsequent reanalysis of Wiles et al.’s (1977) data excluded ex-smokers from the analysis and included five years of follow-up. The resultant cohort consisted of 1393 current smokers and 232 never smokers (Hnizdo, 1992). Previous estimates of dust exposure based on respirable surface area were converted into gh/m^3 for this study. The average duration of underground dust exposure was 24 years and the average concentration of respirable dust was 0.3 mg/m^3. Based on regression models that included dust exposure, height, weight, smoking, and grade of radiologic silicosis as variables, it was estimated that for a 50-year-old miner exposed to the average cumulative dust exposure of 14.4 gh/m^3, declines in FEV_1 and FVC attributable to dust exposure were 236 ml and 217 ml, respectively. The corresponding estimated declines for the highest quartile of exposure, 22.2 gh/m^3, were 364 ml for FEV_1 and 335 ml for FVC. The effects of smoking were somewhat larger than the effects of dust exposure: for a 50-year-old smoker with a 30 pack-year history, the estimated pulmonary function decrements due to smoking were estimated to be 552 ml for FEV_1 and 392 ml for FVC. The effects of smoking and dust exposure on respiratory function were additive. For example, the expected loss of FEV_1 for a 50-year-old smoking miner with a high dust exposure 22.2 gh/m^3 and 30 pack year smoking history was 916 mL. Although data was not provided, the author noted "silicosis was not a significant predictor of any of the measurements of lung function except FEV_1 in non-smokers" (Hnizdo, 1992).

The previous studies had all focused on white South African gold miners, due to the more reliable records available for them. Cowie et al. (1991) undertook the first study of black South African gold miners. They recruited men who underwent routine radiographic surveillance for the detection of silicosis, and selected the sample based on a 5:2 ratio of men with silicosis to those without. The final sample consisted of 857 men with silicosis and 340 men without. Dust exposure was assessed by the duration of underground work in years, and also a categorical intensity score based on occupation type. The presence of silicosis was associated with reduction in all indices of lung function. For example, men with ILO category 2/2 had an FEV_1, FVC, FEV_1/FVC, and MMEF that was 320 mL, 234 mL, 3.3%, and 0.7 L/s lower than men with category 0/0 nodule profusion. When silicosis and smoking were controlled for, the estimated reduction in FEV_1 due to 25 years of dust exposure was 200 ml, and the decline in FEV_1/FVC was 3.3%. The average annual loss in FEV_1 due to dust exposure was 8 ml. The decrease in FEV_1 attributable to smoking one pack-year of cigarettes was estimated to be 6.9 ml (Cowie et al., 1991).

A subset of this cohort (biased towards those with more severe silicosis) was re-evaluated 5 years after the initial cross-sectional assessment (Cowie, 1998). The 242 men who participated had a smaller proportion of never-smokers but did not differ with regards to age or duration of underground mining exposure from the original cohort of Cowie et al. (1991). The subjects were all active miners. Radiographic findings progressed over the follow-up period: 20 out of 59 men whose initial chest radiographs were normal developed radiographic silicosis; in 210 out of the 242 subjects, nodular profusion increased or remained unchanged from baseline; on average, the degree of nodular profusion increased by one subcategory over the follow-up period. Annual declines in FEV_1, FVC, FEV_1/FVC, and diffusing capacity were significantly and directly related to the degree of nodular profusion at baseline. For example, men with category 0, 1, 2, and 3 radiographic profusion at baseline had annual declines in FEV_1 of 37 mL/year, 57 mL/year, 100 mL/year, and 128 mL/year, respectively. As an indication that lung function declines continued even in the absence of further exposure, the 32 men who moved to non-dusty occupations did not differ in their initial FEV_1 or annual decline in FEV_1 from the other 210 men (Cowie, 1998).

In general, the South African gold miner studies observed that pulmonary function declined with increasing cumulative dust exposure, with greater negative effects of exposure occurring in smokers than non-smokers. As well, although the degree of radiographic silicosis correlated to annual declines in lung function (Cowie, 1998), there is some indication that it was cumulative dust exposure, and not the presence of silicosis per se, that affected pulmonary function (Irwig et al., 1978; Hnizdo, 1992).

A recent small Romanian study contradicted some of these conclusions. Subjects were 73 gold miners with silicosis plus 73 miners without silicosis, and each group were of similar age and smoking status (Cocarla et al., 2003). The miners' pulmonary function were evaluated at baseline and after a three year interval by the same team, using the same equipment and under the same test conditions. Area sampling in representative workplaces revealed that mean dust concentrations ranged from 3.3 to 7.3 mg/m^3, with a silica content of 7.9% to 19.2%. Of the 73 miners with silicosis, 18 had PMF and the remainder had either ILO category 1, 2, or 3 nodular profusion. The average decline in FEV$_1$ between the two examinations was 188 mL/year for miners with silicosis compared to 43 mL/year for miners without silicosis. For miners with silicosis, there was no significant difference between the rate of FEV$_1$ decline for smokers (174 mL/year) and non-smokers (202 mL/year). However, for miners without silicosis, the FEV$_1$ decline for smokers (53 mL/year) was significantly greater than for non-smokers (33 mL/year), leading the authors to suggest that dust exposure "masks" the effect of smoking. The miners with silicosis had significantly greater cumulative total dust exposure (87 mg/m^3-years versus 72 mg/m^3-years) than the miners without silicosis. There was a clear dose-response trend between cumulative dust exposure and rate of FEV$_1$ decline in miners with silicosis, but there was no correlation for miners without silicosis. This led the authors to conclude that increased rates of FEV$_1$ decline are dependent on the presence of radiographic silicosis, and not on cumulative dust exposure alone (Cocarla et al., 2003).

The effects of mining dust exposure in the absence of pneumoconiosis was also investigated by Manfreda et al. (1982). They studied 241 men who were randomly selected from 1316 individuals employed in two mining companies in Manitoba, and with the exception of one miner, all radiographs were reported as normal. The miners' pulmonary function data were compared with data derived from an external population sample of 382 men from the same geographic area who were similar to the miners with respect to ethnic origin, age, and smoking status. Underground dust contained 6-9% silica, and approximately 20% of underground dust samples exceeded threshold limit values. Among non-smokers, there were no significant differences between the proportion of workers with abnormal pulmonary function parameters (less than the lower 95% confidence limit of referent values) and the referent population. However, the FVC, FEV$_1$, MMEF, FEF$_{25\%}$, FEF$_{50\%}$, and FEF$_{75\%}$ of smoking miners were 80-90% of the predicted values obtained from the smoking referents, and the differences were significant. The authors concluded that exposure to mining irritants negatively affected pulmonary function, and the effect was predominantly observed in smokers (Manfreda et al., 1982).

Kriess et al. (1989) conducted a population-based prevalence study of respiratory function in a Colorado mining town, 5 months after a temporary lay-off by the major employer, a molybdenum mine. The average silica content of the dust in the mines ranged from 10% to 30%, and limited historical dust sampling revealed that one-quarter of personal samples exceeded 0.1 mg/m^3 respirable quartz, the permissible exposure limit. Out of a census of 1433 eligible adults, 383 males participated, of which 62% had worked at the mine and had some dust exposure. In a multiple regression analysis controlling for age, height, and smoking, cumulative dust exposure was associated with decreased FEV$_1$, FEV$_1$/FVC, MMEF, and FEF$_{75\%}$. When the analysis was restricted to 132 never-smokers, dust exposure was associated with a decline in flow rates and lung volumes, and an increase in diffusing capacity. Dust exposure alone had similar quantitative effects as smoking alone on FEV$_1$, FEV$_1$/FVC, and MMEF. Dust exposure combined with smoking was associated with greater airflow limitation than smoking alone. The lower expiratory flow rates in non-smokers were attributed to their achievement at lower lung volumes, as flows compared at absolute lung volumes were increased for non-smokers. The overall conclusion was that mining exposure was associated with minor pulmonary function changes in non-smokers and aggravation of hyperinflation and airflow limitation in smokers (Kriess et al., 1989).

2.2.2.2 Other Occupational Groups

In addition to studies of miners, numerous other occupational groups have been studied to explore the relationship between silica exposure and pulmonary function, including granite workers, foundry workers, construction workers, firebrick workers, and tunnel workers.

Malmberg et al. (1993) performed a small longitudinal study of granite crushers who were exposed to average concentrations of respirable silica of 0.2 mg/m^3, twice the occupational exposure limit. Workers and male population referents matched for age and smoking were examined in 1976 and 1988. Of the 62 workers who participated in the 1976 examination, 45 returned for assessment in 1988. The same technicians and type of testing equipment were used in both surveys. Only one granite crusher had radiographic silicosis, and the remainder had normal chest x-rays. In 1988, the granite crushers had significantly lower FEV$_1$/VC (73% versus 76%) and FEF$_{50\%}$ (4.5 L/s versus 5.1 L/s) as compared to the referents. Over the 12-year follow-up period, FEV$_1$, FEV$_1$/FVC, maximal expiratory flow, and FEF$_{50\%}$ decreased by 5%, 5%, 8%, and 14% more, respectively, in the granite crushers as compared to the referents. Retired workers tended to have slightly greater lung function changes than active workers, suggesting that lung function changes due to silica exposure may continue after exposure has ceased. Five granite crushers but no referents had FEV$_1$ values less than 80% of predicted, all of who were either smokers or ex-smokers; with the exception of one of these workers, however, silica exposure had been low. The authors concluded that overall effects of silica exposure on lung function were modest and that exposure may interact with smoking to aggravate functional changes (Malmberg et al., 1993).

Another small study examined 153 granite quarry workers without radiographic pneumoconiosis, who had a FEV_1/FVC ratio of greater than 0.75 and a FVC of greater than 75% of predicted (Chia et al., 1992). Average quartz content in respirable dust was 28%, and workers were divided into low, medium, and high exposure based on job type. After adjustment for age, height, and smoking status, FEV_1 and FVC were not significantly different across exposure groups, but $FEF_{75\%-85\%}$, and $FEF_{75\%}$ were significantly lower in higher exposure groups as compared to lower exposure groups ($FEF_{75\%-85\%}$: 1.3 L/s in the low exposure group versus 0.8 L/s in the high exposure group; $FEF_{75\%}$: 1.9 L/s versus 1.2 L/s). When the study group was divided into smokers and non-smokers, smokers had a greater degree of airways obstruction than the non-smokers, but the trend of greater small airways obstruction in the higher exposure groups were still observed, especially for $FEF_{75\%-85\%}$ and $FEF_{75\%}$. Because maximum flow at small lung volumes reflected the function of the small airways, it was concluded that silica exposed workers had small airways obstruction in the absence of radiological silicosis or large airways obstruction (Chia et al., 1992).

A series of studies of Vermont granite workers did not find evidence for an adverse effect of silica dust exposure on pulmonary function (Graham et al., 1981; Graham et al., 1994). In the earlier study, granite shed workers who had undergone pulmonary function testing over three separate time periods (1970, 1974, and 1979) were examined longitudinally. For 402 workers studied, the volume of FEV_1 loss over the first 4-year time period was 169 mL, whereas from 1974-1979, the average FEV_1 value was essentially the same as the 1974 average value. When the analysis was restricted to 242 workers with 20 or more years of employment in the granite sheds, the loss of FEV_1 from 1974-1979 averaged 7 mL per year. A follow-up report with eight additional years of follow-up did not find an association between years of granite exposure and declines in FEV_1 or FVC (Graham et al., 1994).

A subsequent study addressed the possibility of survivor bias in the previous granite shed studies by analyzing respiratory function data from 618 white male workers who started work after 1940, had no previous dusty trade experience, and were 25-65 years of age in 1970 (Eisen et al., 1995). These workers provided up to five follow-up pulmonary function measurements after their initial 1970 survey. The only outcome studied was FEV_1, with expected values calculated form the 1976 prediction equations of Knudson et al. (1976). Dust exposures were determined from personal sample data collected in 1970 and 1976, and average annual dust levels were estimated for every job in the industry after adjusting for the effects of season, survey year, job, and granite shed. The cohort was divided into two groups, those with a valid FEV_1 measurement in the final survey conducted in 1975 (survivors), and those without (dropouts). Reasons for missing FEV_1 values included termination, non-participation, or test failure. The survivors and dropouts were similar based on 1970 information on age, height, cumulative smoking, and exposure. Past dust exposures averaged 6.72 and 6.94 mg/m^3 – years for the dropouts and survivors, respectively. The dropouts lost FEV_1 volume at an average rate of 69 mL/year whereas the survivors lost 44 mL/year. Greater rates of FEV_1 decline were seen in dropouts as compared to survivors across all smoking categories. Among the survivors, annual change in FEV_1 was associated with height and smoking but not current dust exposure. However, for the dropouts, the effect of current dust exposure was almost three times as great and of borderline significance, suggesting that dropouts had an additional 100 mL annual loss of FEV_1 beyond that due to smoking or age for each additional mg/m^3 of current granite dust exposure. When lifetime exposure was considered, a 4 mL per year loss of FEV_1 was associated with each additional mg/m^3 of dust exposure for dropouts, whereas the survivors demonstrated a non-significant positive relationship between FEV_1 and dust exposure. The authors concluded that their examination of survivors produced results that were similar to earlier studies. However, such results were not representative of all granite workers, as differential effects of dust exposure were apparent between survivors and dropouts (Eisen et al., 1995).

Studies of workers exposed to silica in other industries also demonstrate adverse effects on pulmonary function from silica exposure. A South Korean cross sectional study compared 209 male foundry workers exposed to silica dust with 239 male office workers, and excluded those subjects with a history of pulmonary tuberculosis, chronic bronchitis, emphysema, or asthma (Koo et al., 2000). There were no significant differences between the two groups with regards to age, height, weight, and pack-years among smokers. Details regarding past occupational exposures and subject recruitment methods were not provided. Pulmonary function indices were expressed as a percentage of normal values, based on equations published by Knudson et al. (1976). Cumulative dust exposure was defined as the product of quartz content, determined from personal dust samples, and exposure duration. Mean quartz concentrations ranged from 0.023 to 0.079 mg/m^3, and the maximal concentration was 0.147 mg/m^3. With the exception of FVC, all ventilatory indices were significantly lower in the foundry workers as compared to the office workers. For example, FEV_1, FEV_1/FVC, and MMEF were 3.17 L, 75%, and 2.92 L/s, respectively, in foundry workers versus 3.56 L, 83%, and 4.13 L/s for the corresponding indices in office workers. However, the percent predicted values in the foundry workers were well within the normal range, with FVC, FEV_1, and FEV_1/FVC at 101%, 95%, and 101% of predicted, respectively. When lung function indices were stratified across cumulative dust exposure levels (<0.3, 0.3-9.9, >9.9 mg/m^3-years), there was no indication of a dose-response relationship for FEV_1, FVC, or FEV_1/FVC. Although not significant, MMEF and maximal expiratory flows at 25%, 50% and 75% of FVC all tended to decrease with increasing cumulative dust exposure category (Koo et al., 2000).

In another recent study of foundry workers, Hertzberg et al. (2002) evaluated the effectiveness of current occupational exposure limits for respirable silica dust in workers without radiographic silicosis. They studied 1028 current and former foundry employees who had annual pulmonary function tests, 523 of whom had one or more acceptable tests, information on smoking status, and information on dust exposure. Dust exposure measurements were available for almost 90% of the years covered by the study. Among non-smokers, there was no association between cumulative silica exposure and FEV_1, FVC, or FEV_1/FVC. However, for smokers, there were significant associations between increasing cumulative silica exposure and decreasing FEV_1, FVC, and FEV_1/FVC. A multiple linear regression analysis with cumulative silica exposure, pack-years of cigarette smoking, ethnicity, age, and height as independent variables demonstrated highly significant relationships between cumulative silica exposure and each of the three pulmonary function end points. Estimated longitudinal declines for FEV_1 and FVC were 1.1 mL/year and 1.6 mL/year, respectively, for each mg/m^3 of silica exposure, after adjusting for ethnicity and pack-years smoked. Based on the regression results, a worker exposed to the current United States Occupational Safety and Health Administration occupational exposure limit for silica of 0.1 mg/m^3 for 20 years would have a silica exposure attributable reduction in FEV_1 of 52 mL, in FVC of 69 mL and in FEV_1/FVC of 0.75%; for workers exposed for 40 years at the same level, the respective reductions would be 104 mL, 138 mL, and 1.49%. Corresponding values for smoking one pack of cigarettes per day for 40 years were a loss in FEV_1 of 312 mL, in FVC of 232 mL, and in FEV_1/FVC of 3.4% (Hertzberg et al., 2002).

A Norwegian study of tunnel workers was one of the few studies to evaluate a number of occupational exposures concurrently, including mixed dust, alpha quartz (crystalline silica), oil mist, nitrogen dioxide, and products of diesel combustion (Ulvestad et al., 2001). Tunnel workers, outdoor construction workers, and white-collar construction workers from the same construction company were examined in 1991, and 83% of the original cohort of 417 men participated in a second examination in 1999. Personal exposure measurements were conducted between 1996 and 1999 and were considered to be representative of the period of study. Average exposure to respirable alpha quartz ranged from 0.019 mg/m^3 in shotcreters to 0.044 mg/m^3 in drillers. There were no significant differences between the occupational groups with regards to age, height, atopy, and duration of employment. None of the workers had any radiographic evidence of pneumoconiosis. There were no associations between pre-1991 exposures and decreases in lung function. During the 8-year study period, however, decreases in FEV_1 were significantly associated with cumulative exposure to respirable dust and α-quartz. Predictions from regression models generated estimates for annual FEV_1 declines in a 40-year-old worker were: 25 ml in a non-exposed non-smoker, 35 ml in a non-exposed smoker, 50 ml in a non-smoking driller, and 63 ml in a non-smoking shotcreter.

Meijer et al. (2001) compared 144 four concrete workers at two factories with 110 factory workers who produced office equipment. Silicosis was not observed in any worker from periodic radiographs performed in the years before the study. Dust measurements were obtained from personal samplers, and a questionnaire assessed occupational history, smoking habits, and respiratory symptoms. As determined by personal dust samples, the overall average dust concentration was 0.77 mg/m^3 and the average respirable silica concentration was 0.059 mg/m^3. Average cumulative silica dust exposure was 0.50 and 0.63 mg/m^3–years, respectively for the two concrete plants included in the study. After controlling for smoking habits and allergic history, FEV_1, MMEF, and FEV_1/FVC were all negatively associated with dust exposure, although a dose-response relationship was not apparent. The magnitude of the association was small, however, and mean values for FEV_1, FVC, and FEV_1/FVC in concrete workers were all 98% to 112% of predicted (Meijer et al., 2001).

A large Dutch study compared 1335 actively employed construction workers (32% of the invited study population) with a Dutch referent population (Tjoe-Nij et al., 2003a). Dust measurements were only available for a subset of job categories, and the expert opinions of three industrial hygienists were used to classify 36 different jobs on a 10-point exposure scale, which formed the basis of a cumulative exposure index (an accompanying paper (Tjoe-Nij et al., 2003b) had observed that pneumoconiosis prevalence increased with increasing cumulative exposure index). There was a large variability of exposure, ranging from 0.0016 to 4.7 mg/m^3 respirable quartz, with arithmetic and geometric means of 0.40 and 0.09 mg/m^3, respectively. After adjustment for smoking, FEV_1 and FVC were 120 mL and 130 mL lower, respectively, in the construction workers as compared to the referents. Thirty-seven workers had radiographic pneumoconiosis (ILO category 1/1 or greater); the presence of pneumoconiosis was associated with 267 mL lower FEV_1,181 mL lower FVC, and a 2.5% reduction in FEV_1/FVC. With the exception of FVC (- 5 mL per year exposed), neither the cumulative exposure index nor duration of exposure was associated with lung function (Tjoe-Nij et al., 2003a)

In addition to studies of silica-exposed worker groups, population-based studies have suggested an association between silica exposure and impaired lung function. Humerfelt et al. (1998) performed a large cross sectional community survey that consisted of a questionnaire, chest radiograph, and spirometry. All men living in western Norway and born between 1944 and 1958 were invited to participate. Predicted maximum spirometry values were calculated from linear regression models using 3027 healthy asymptomatic never smokers without occupational quartz exposure. Of the 45380 eligible subjects, 26106 completed the self-administered questionnaire, had successful spirometric tests, and had normal chest radiographs. Of these men, 3425 had previous exposure to quartz. The FEV_1/FVC ratio was lower among the exposed subjects (79.1% versus 79.6%) after adjusting for age and smoking. Among exposed workers, the ratio decreased with increasing years of exposure and the trend was significant.

Estimated FEV_1 declines were 6.9 mL for someone smoking 20 cigarettes per day for one year, and 4.3 mL for each year exposed to quartz. Although this study benefited from large subject numbers, quartz exposure and possible confounding exposures could not be directly assessed.

Firebrick manufacturing workers were examined in a cross-sectional study that compared them with administrative workers (Liou et al., 1996). After workers with dust, gas, or fume exposure from previous occupations and those with a history of chronic obstructive pulmonary disease prior to working in the firebrick plant were excluded, 526 manufacturing workers and 164 administrative workers remained in the study. Seven percent of manufacturing workers and 0.6% of administrative workers had radiographic pneumoconiosis, and there was a dose-response relationship between duration of employment and pneumoconiosis, with 13% of workers employed more than 10 years having category 1 or 2 pneumoconiosis versus 2% of workers with less than 5 years employment duration. There was no difference in FVC between the manufacturing and administrative workers, but FEV_1/FVC, MMEF, $FEF_{50\%}$, and $FEF_{75\%}$ were significantly lower in the manufacturing workers. These differences persisted when the analysis was stratified by smoking status. After adjustment for smoking, significant dose-response relationships were observed between duration of employment and FEV_1/FVC, MMEF, $FEF_{50\%}$, and $FEF_{75\%}$. However, these parameters were well within the normal range based on percent-predicted values. For example, in the highest exposure group (greater than 10 years employment), the percent-predicted values for FEV_1, FVC, and MMEF were 104%, 107%, and 87%, respectively, while the average FEV_1/FVC in this group was 82.3%. In a two-year follow-up study of 291 manufacturing workers and 72 administrative workers, there were no significant differences in FVC or FEV_1 decline between the two groups, after adjusting for age, sex, height, and smoking status (Chen et al., 2001). However, declines in FEV_1/FVC, peak expiratory flow rate, MMEF, and $FEF_{50\%}$ were significantly greater in the firebrick workers as compared to the controls, but the absolute differences were modest.

2.2.3 Influence of Radiographic Pneumoconiosis

Several studies have explored the nature of the relationship between silica exposure, pulmonary function, and the presence of radiographic pneumoconiosis. Some studies of South African gold miners have determined that it is the degree of dust exposure and not silicosis per se that is a significant predictor of lung function decline (Hnizdo, 1992; Irwig et al., 1978). Some recent studies have suggested that dust exposure in the absence of silicosis does not lead to adverse changes in lung function (Cocarla et al., 2003; Tjoe-Nij et al., 2003a), however there are an abundance of studies that have observed subtle lung function decrements in the absence of radiographic pneumoconiosis (Chia et al., 1992; Hertzberg et al., 2002; Humerfelt et al., 1998; Malmberg et al., 1993; Manfreda et al., 1982; Meijer et al., 2001; Ulvestad et al., 2001). A recent review also concluded that silica dust exposure at levels that are not associated with radiological silicosis can lead to the development of chronic bronchitis, emphysema, and small airways disease, resulting in airflow obstruction (Hnizdo et al., 2003).

The pulmonary function changes observed in the absence of radiologic silicosis are relatively small. However, as silicosis severity increases, as determined by increasing levels of nodular profusion and/or the development of complicated silicosis (ie PMF), more marked declines in pulmonary function become evident. This has been demonstrated in studies of South African gold miners, discussed above (Cowie et al., 1991; Cowie, 1998). Additional support comes from studies of granite quarry workers by Ng and colleagues. In the first study, 73 granite quarry workers with silicosis had periodic radiographic and lung function examinations between 1975 and 1981 (Ng et al., 1987). At the initial examination, 73% of subjects had simple silicosis, and the remainder had complicated silicosis (presence of large opacities, tuberculosis, pleural thickening, eggshell calcification, calcified small opacities, enlarged hilar lymph nodes, or emphysema). At baseline, subjects with complicated silicosis had lower FVC and FEV_1 (values approximately 85% of predicted) than subjects with simple silicosis (values approximately 95% of predicted). Mean follow-up time was 7.2 years and half of the subjects had radiologic progression (incremental change in ILO grade). On average, declines in FEV_1 and FVC were 79 mL/year and 75 mL/year, respectively, in subjects with simple silicosis and 84 mL/year and 88 mL/year, respectively, in subjects with complicated silicosis. After adjusting for baseline values, age, and smoking, those with radiological progression had significantly greater decrements in both parameters than those without progression.

A subsequent study included 82 past workers who were not known to be silicotic at their termination, 50 past workers with silicosis, and 206 current workers (Ng et al., 1992). After adjustment for age, height, cigarette-years, and cumulative silica exposure, FEV_1, FVC, and FEV_1/FVC were not significantly different between the workers without radiographic opacities (ILO grade 0/0) and workers with category one profusion (ILO grade 0/1 to 1/2). However, these pulmonary function parameters were significantly lower in subjects with profusion graded 2/1 or higher. In multiple regression analyses adjusted for age, height, and cigarette-years, both cumulative silica exposure and profusion of small opacities were individually associated with decrements in FEV_1 and FVC. However, there was no additional effect of cumulative silica exposure when profusion of small opacities was simultaneously allowed for. The authors concluded that inhalation of respirable silica did not lead to lung function loss if the dust exposure was not sufficient to produce lung fibrosis. But for individuals with nodular profusion graded as 2/1 or higher, the observed lung function loss was attributable to fibrotic disease (Ng et al., 1992).

These conclusions were supported by an earlier study of 94 workers evaluated for possible compensation (Begin et al., 1988). The subjects were granite workers, foundry workers, and gold miners, and 90% of them were current or former smokers. Subjects were divided into 4 roughly equivalent-sized groups: group 1 did not meet diagnostic criteria for silicosis, group 2 had simple silicosis, group 3 had simple silicosis on chest radiograph but conglomeration and/or coalescence on CT scan (complicated silicosis), and group 4 had complicated silicosis on both chest radiograph and CT scan. Pulmonary function parameters were within normal prediction ranges for subjects in group 1. Total lung capacity, FVC, diffusing capacity, FEV_1, FEV_1/FVC, MMEF, and $FEF_{50\%}$ all decreased incrementally between the 4 groups, with the lowest values occurring in group 4. In this study, disease severity as defined radiographically by CT scan was associated with both restrictive lung function changes and airflow limitation.

However, subsequent studies have concluded that although lung function is impaired with increasing degrees of silicosis, it is the presence of emphysema, independent of silicosis, that is responsible for the observed pulmonary function decline. In a study by Genevois et al. (1998), 35 silica-exposed subjects underwent computed tomography scans and pulmonary function tests. All subjects had either low-grade pneumoconiosis (ILO grade less than 1/1) or had well-established pneumoconiosis but no PMF. There were significant associations between the extent of emphysema and measured pulmonary function parameters, but there were no significant differences between workers with micronodules and those without with regards to the extent of emphysema or pulmonary function results. This study concluded that exposure to silica dust could adversely affect pulmonary function, but that this was independent of pneumoconiosis, as detected by radiographic micronodules on CT scans (Genevois et al., 1998).

A study that utilized plain chest x-rays arrived at similar conclusions. Wang et al. (1999b) studied 220 workers from a firebrick production plant who had at least three years of silica exposure. Half of the workers had radiographic silicosis and half did not. Workers were evaluated every 2 to 3 years, and their most recent chest radiograph was evaluated for the presence of hyperinflation, as a marker of emphysema. The prevalence of respiratory symptoms and radiographic hyperinflation increased significantly with higher silicosis categories. FVC, FEV_1, and FEV_1/FVC significantly decreased with silicosis category. In a regression analysis, silicosis severity was significantly associated with FEV_1, FVC, FEV_1/FVC, and diffusing capacity. However, these significant relationships were no longer apparent when hyperinflation was added to the model. Radiographic hyperinflation was significantly associated with a decrease in FEV_1 and FEV_1/FVC. When smokers and non-smokers were compared separately, workers with hyperinflation had a lower mean value for each pulmonary function parameter than those without hyperinflation, regardless of silicosis. The authors concluded that pulmonary function impairments in silica-exposed workers were likely related to emphysema, and not silicosis (Wang et al., 1999b).

These results were similar to those of Cowie et al. (1993), who used CT scans to study a subset of 70 male South African gold miners selected from the 1197 man cohort studied by Cowie et al. (1991). Fifteen of these men did not have silicosis, and 13 of the 55 with silicosis had large opacities (PMF). Emphysema was present in 48 men, and smokers were also more likely to have emphysema than non-smokers. After controlling for smoking and duration of underground mining work, silicosis was significantly associated with reductions in FEV_1/FVC and MMEF. However, after emphysema was included in the model, there was no association between silicosis and lung function parameters. After controlling for silicosis, years worked underground, and smoking, emphysema was associated with reductions in FEV_1, FEV_1/FVC, MMEF, and diffusing capacity.

In a smaller study of 30 compensation subjects, Kinsella et al. (1990) used CT scans to evaluate 12 non-smokers and 18 smokers with silicosis. Nodular profusion was graded and dichotomized into subjects without nodular confluence and those with confluence of nodules (PMF). In subjects with mild silicosis, there were significantly more smokers than non-smokers with emphysema, but there was no difference in percent emphysema between smokers and non-smokers with nodular confluence. Increasing severity of emphysema was significantly associated with lower diffusing capacity, FEV_1, and FVC. In a multiple linear regression analysis, percent emphysema and silicosis grade were both independently associated with diffusing capacity, but only percent emphysema was independently associated with FEV_1 or FVC. It was concluded that it was primarily the degree of emphysema, not silicosis, which affected pulmonary function and that silicosis did not cause significant emphysema in the absence of nodular confluence.

A recent review of the relationship between silicosis and lung function concluded that there is no distinguishable loss of lung function with ILO category 1 profusion, with slight decreases noted for category 2, and the greatest losses observed in category 3 silicosis and PMF (Gamble et al., 2004). Although simple silicosis does not appear to be a major risk factor for emphysema in the absence of smoking, emphysema is more common in the presence of PMF. Although relatively few studies have examined silicosis, emphysema, and dust exposure simultaneously, in studies that have, it appears that pulmonary function is more closely related to emphysema, rather than silicosis (Gamble et al., 2004).

2.2.4 Influence of Smoking

In general, the studies summarized above indicate smoking has a greater negative effect on pulmonary function than silica exposure alone. Hnizdo (1992) estimated that after several decades of exposure to the highest quartile of dust exposure in gold miners, FEV_1 and FVC would decline by the 364 and 335 mL, respectively. This effect of high cumulative dust exposure was still less than the estimated losses for a 30 pack-year smoking history: 552 mL for FEV_1 and 392 mL for FVC. In their study of foundry workers, Hertzberg et al. (2002) found that pulmonary function losses from a 40-year career exposed to respirable silica at 0.1 mg/m^3 (104 mL drop in FEV$_1$ and 138 mL drop in FVC) were considerably lower than the expected decline from a 40 pack-years of smoking (312 mL for FEV$_1$ and 232 mL for FVC). A large Norwegian population study estimated that a year of smoking one pack per day would result in a 6.9 mL FEV$_1$ decline, versus a 4.3 mL decline for each year exposure to quartz dust (Humerfelt et al., 1998). The annual decline estimates of Cowie et al. (1991) were in opposition to other studies, in that their estimate for annual FEV$_1$ decline attributable to dust exposure exceeded the estimate for smoking (8 mL versus 6.9 mL). In only one study was it concluded that dust exposure "masked" the effects of smoking (Cocarla et al., 2003), however this observation was only made in miners with very heavy cumulative dust exposure and a relatively significant degree of silicosis.

The overall weight of evidence indicates that any adverse effects of silica exposure on pulmonary function are more marked in smokers than in non-smokers (Chia et al., 1992; Hnizdo et al., 1990; Kriess et al., 1989; Malmberg et al., 1993; Wiles et al., 1977). The relationship of the combined effects of smoking and silica exposure has been observed to be additive (Hnizdo, 1992) and may also be multiplicative (Hnizdo et al., 1990). In studies that have stratified their analyses in order to examine non-smokers only, many have not observed any adverse effects of dust exposure on lung function (Hertzberg et al., 2002; Manfreda et al., 1982; Ulvestad et al., 2001), whereas others have noted subtle changes only (Chia et al., 1992; Kriess et al., 1989).

Several mortality studies support the conclusion that smoking is more potent than silica exposure with respect to adverse effects on the lungs. One study (Hnizdo, 1990) looked specifically at mortality from chronic obstructive pulmonary disease disease (COPD) in relation to dust exposure in the original group of 2209 miners studied by Wiles et al. (1977). Of the 794 miners who had died and undergone autopsies, 66 were classified as having died from COPD (which included the International Classification of Diseases codes for bronchitis, emphysema, and chronic airways obstruction). A nested case-referent design was used, with six age-matched miners selected at random for every case of a miner who died of COPD. There was a dose-response relationship between both dust exposure and amount smoked and risk of death from COPD. Odds ratios and 95% confidence intervals for increasing dust exposure categories increased from 2.5 (0.9, 6.9) for the second-lowest exposure category to 5.3 (1.8,15.9) for the highest exposure category. Odds ratios for cigarette smoking ranged from 8.0 (0.9, 69.7) for 16 years of smoking up to 32.3 (4.2, 248.2) for 34 years of smoking. For those in the highest categories of dust and smoking exposure, the combined effects of the two exposures were more than additive but less than multiplicative. Based on attributable risk calculations, the estimated proportion of cases of death due to COPD caused by dust, smoking, and dust and smoking combined were 5%, 34%, and 59%, respectively (Hnizdo, 1990).

In a larger mortality study, Reid et al. (1996) followed a cohort of almost 5000 white South African gold miners, who were 39 to 54-years-old at inception, for 20 years. Cause of death was determined by International Classification of Diseases coding on death certificates, and the total white South African male population was the reference population. A case-control analysis was also performed matching cases who died from COPD, lung cancer, or ischaemic heart disease (IHD) to miners born in the same year as the case and who outlived the case. The total number of deaths in the cohort numbered 2032, which resulted in a significantly elevated standardized mortality ratio (SMR) of 130 for death from all causes. Several specific causes of death had elevated SMRs, including lung cancer, COPD, pneumoconiosis, IHD, tuberculosis, liver cirrhosis, and renal failure. In the case control analysis, smoking had significantly elevated odds ratios for lung cancer, COPD, and IHD. There were no COPD deaths in non-smokers. Years spent working underground was not a risk factor for COPD death. However, cumulative dust exposure was associated with death from COPD in smokers, such that for a typical miner's dust exposure over a career, the risk of COPD was double that of smoking alone. The overall conclusion was that underground mining and dust exposure contributed to the development of COPD, but that further insult from smoking was required to develop disease. Overall, the elevated SMR's in this cohort were attributed to the unhealthy lifestyles of the miners, which included smoking and heavy alcohol consumption (Ried et al., 1996).

In another large autopsy study of 1553 subjects, there was a consistent association between silica dust exposure from 35 years of age and the presence of emphysema. Estimates for the odds of emphysema for a miner exposed to high-dust occupations for 20 years ranged from 2.1 to 3.5, depending on the exposure estimate used. In this study, only 4 miners had emphysema out of 163 non-smokers, and no association was found between emphysema score and dust exposure among the non-smokers. It was concluded that the risk of significant emphysema associated with dust exposure was specific to smokers only (Hnizdo et al., 1991).

A subsequent study limited the analysis to 242 lifetime non-smokers, of whom 174 had lung function tests performed at the Medical Bureau for Occupational Diseases prior to their death (Hnizdo et al., 1994). An insignificant degree of panacinar emphysema was found, with only four subjects classified as having a moderate degree of emphysema: two of these subjects had normal lung function, one had coronary heart disease, and one had pulmonary tuberculosis and only one year of underground mining experience. The degree of emphysema was not a predictor of the most recent lung function measurements, nor was it associated with years of gold mining, cumulative dust index, or parenchymal silicosis. It was concluded that silica dust exposure was unlikely to cause a moderate degree of airflow limitation in non-smokers (Hnizdo et al., 1994).

2.2.5 Silica Summary

Data on the effects of silica exposure on lung function have been obtained from a variety of occupational exposure groups. Because of differences in study design, subject selection, exposure source, exposure levels, and other factors, outcomes and conclusions vary across studies. Nevertheless, certain general trends are apparent.

A reduction in FVC, suggestive of restrictive disease, has been observed in some studies (Hnizdo et al., 1990; Hnizdo, 1992; Tjoe-Nij et al., 2003a) but not others (Chia et al., 1992; Irwig et al., 1978; Koo et al., 2000; Liou et al., 1996; Malmberg et al., 1993). A reduction in FVC has been noted in studies of miners with advanced silicosis (Cowie et al., 1991, Cowie, 1998), or as an observation in smokers only (Hertzberg et al., 2002; Manfreda et al., 1982).

Typically when pulmonary function abnormalities have been observed, they are reflected in measures representative of airflow in large (eg FEV1, FEV_1/FVC) and small (eg MMEF, $FEF_{75\%}$) airways. Declines in FEV_1 and FEV_1/FVC have frequently been observed, often in a dose-response fashion with increasing cumulative silica exposure (Cowie, 1998; Eisen et al., 1995; Hertzberg et al., 2002; Hnizdo, 1992; Humerfelt et al., 1998; Irwig et al., 1978; Tjoe-Nij et al., 2003a; Ulvestad et al., 2001), and often in combination with declines in measures of small airways function, such as MMEF (Cowie et al., 1991; Hnizdo et al., 1990; Koo et al., 2000; Malmberg et al., 1993; Manfreda et al., 1982; Meijer et al., 2001; Wiles et al., 1977). In a few studies, changes in markers of small airways function have been the only observed abnormalities associated with silica exposure (Chen et al., 2001; Chia et al., 1992; Liou et al., 1996; Kriess et al., 1989).

The declines in pulmonary function associated with advanced silicosis can be substantial, with declines in FEV_1 in the range of 128 mL/year to 188 mL/year (Cowie, 1998; Cocarla et al., 2003). In the absence of advanced silicosis, however, reductions in FEV_1 associated with silica exposure are more modest, in the area of a 100 to 300 mL loss of FEV_1 over a working career (Cowie et al., 1991; Hertzberg et al., 2002; Hnizdo, 1992).

Clinically significant adverse effects of silica exposure on pulmonary function are usually only observed in smokers. In the absence of advanced silicosis, pulmonary function in non-smokers is usually not affected to any great degree.

2.3 Asbestos

Asbestos refers to a group of hydrated silicate fibres that are classified into six types based on different chemical and physical features. These six types can be broadly classified in two main groups: serpentine fibres (chrysotile) and amphibole fibres (crocidolite, amosite, anthophyllite, actinolite, and tremolite). Because of the physical and chemical properties of asbestos (eg. high tensile strength, chemical resistance, heat resistance), it is used in a wide variety of industrial products, including insulation, textiles, cement, friction products such as brake linings, and construction materials. Specific trades at high risk for asbestos exposure include insulators, sheet metal workers, plumbers and pipefitters, steamfitters, boilermakers, and numerous shipboard trades (Brodkin et al., 2005).

The relationship between inhalation of asbestos fibres and respiratory disease has been recognized for decades. Asbestos-related malignancies include bronchogenic carcinoma and malignant mesothelioma (which may also arise in extrapulmonary sites). Parenchymal fibrosis (asbestosis), pleural fibrosis (diffuse pleural thickening), pleural plaques (circumscribed pleural thickening), rounded atelectasis, and benign exudative pleuritis (pleural effusion) are examples of non-malignant asbestos-related pulmonary diseases (Brodkin et al., 2005; Manning et al., 2002).

Asbestos fibres are easily respirable, and their ability to cause disease is greatly influenced by fibre diameter and length. Fibres less than 3 μm in diameter can penetrate cell membranes and translocate to the interstitium of the lung, contributing to asbestos diseases. Fibres shorter than 3 μm in length are phagocytosed by macrophages and removed via the lung lymphatic system, whereas fibres longer than 5 μm cannot be completely phagocytosed, stay in the tissues longer, and sustain the cascade of cellular and inflammatory events necessary for the pathogenesis of asbestos diseases (Begin et al., 1996). Current evidence indicates that the amphibole asbestos fibres are more potent than chrysotile in causing disease, and this is believed to reflect the greater biopersistence in the lungs of the amphibole fibres (Manning et al., 2002; Mossman et al., 1998).

The initial site of injury following asbestos dust inhalation is the bifurcation of alveolar ducts, where the terminal bronchioles divide into individual alveolar spaces of the lung. Alveolar epithelial cells take up the asbestos fibres; the piercing of the alveolar wall by the fibres causes cellular injury and is the major route for fibres to reach the interstitium. The damaged cells and the presence of asbestos fibres causes the accumulation of alveolar macrophages, which leads to a release of inflammatory mediators and subsequent cascade of events, eventually leading to a fibrotic tissue response. Tissue damage is caused directly by asbestos fibres via the generation of reactive oxygen intermediates (resulting in injury to intracellular macromolecules and associated lipid peroxidation), and indirectly via activated macrophages that secrete cytokines and other inflammatory mediators that enhance tissue injury (Begin et al., 1996; Brodkin et al., 2005).

Inflammation and fibrosis occur in a dose-response fashion after inhalation exposure to asbestos. With lower exposures, the inflammatory lesions are reversible and histological examinations indicate that there is no evidence of asbestosis at low exposure levels. However, with higher exposures, intense and protracted inflammatory changes result in cell proliferation and excessive deposition of collagen and other extracellular matrix components, eventually leading to the clinical presentation of asbestosis (Mossman et al., 1998). There is evidence that there is a threshold fibre dose below which asbestosis is not observed, likely in the range of 25 to 100 fibres/mL-year (Mossman et al., 1998). It is estimated that at an airborne asbestos exposure level of 0.1 fibres/mL over a working career would result in an excess lifetime risk of asbestosis of 2 in 1000 (Brodkin et al., 2005). The latency from onset of asbestos exposure to the appearance of asbestosis is dependent on the exposure level, from a mean of about 5 years in past studies, secondary to extremely high exposures, to a duration of exposure of 12 to 20 years in more recent studies (Mossman et al., 1998).

The pattern of pulmonary function abnormalities associated with asbestosis is classically described as restrictive; typical findings include decreased FEV_1, FVC, TLC, and RV with a maintained FEV_1/FVC ratio. In addition to restrictive changes associated with parenchymal effects of asbestosis, pathophysiologic changes in small airways can cause airflow obstruction, and diminished mid-expiratory flow rates (eg MMEF) appear to be the most sensitive PFT parameter for assessing early obstructive changes among asbestos-exposed workers (Brodkin et al., 2005)

2.3.1 Historical Studies

The physiological consequences of asbestos exposure, measured through pulmonary function testing, have been the subject of a large body of research dating back to the 1940's. In a review of studies from the 1940's to the early 1970's, Miller (1993) noted that the pulmonary function abnormalities observed in asbestos-exposed workers were consistent with interstitial lung disease, such as hyperventilation, reduced vital capacity, oxygen desaturation with exercise, reduced pulmonary compliance, and reduced diffusing capacity.

In one of the largest early reviews of asbestos-exposed workers, Leathart (1968) found that of 181 workers studied, there was considerable overlap of measured parameters between individuals with certified asbestosis and those who were exposed but did not have asbestosis. However, diffusing capacity and dynamic compliance were significantly different between workers with and without asbestosis. In a smaller group of workers observed for 2 to 9 years, progressive loss of lung function occurred in the absence of further asbestos exposure;

compliance, vital capacity, and diffusing capacity declined an average of 19%, 16%, and 26%, respectively, over the observation period.

In an attempt to identify early pulmonary function markers of asbestos-related effects prior to the development of asbestosis, Jodoin et al. (1971) studied 24 chrysotile asbestos miners and millers with normal chest radiographs. Differences in pulmonary function parameters between the two groups were small except for static pulmonary compliance, which was significantly lower in the group with greater exposure. Unlike vital capacity and diffusing capacity, only compliance showed a consistent relationship with the level of dust exposure. The authors concluded that exposure to asbestos dust affected the mechanical properties of the lungs in the absence of radiographic changes and before other measurements of lung function were generally affected.

Studies of the relationship between asbestos exposure and pulmonary function from the 1970's onward have been of two main types: i) studies of workers in specific industries (eg mining and milling, asbestos-cement manufacturing), which have often included some measurement of asbestos exposure, and ii) studies of workers in specific trades (eg insulators, sheet metal workers), with subjects recruited through unions. The latter type benefit from large subject numbers but are limited by poor asbestos exposure assessment, which is usually characterized by duration of time spent in a specific trade.

2.3.2 Studies of Workers in Specific Industries

In a cross-sectional study, Weill et al. (1975) examined 859 workers at two asbestos-cement manufacturing plants, with 91% participation. Dust exposures were predominantly chrysotile asbestos, with smaller proportions of amosite and crocidolite. The cement also contained high amounts of silica. Total dust exposure, expressed as million particles per cubic feet of air times years (mppcf-yrs), was determined by multiplying dust sampling results for each job type by the time spent in each job and divided into five exposure categories, ranging from <50 mppcf-yrs to >400 mppcf-yrs (1 mppcf-yr = 35.31 million particles/m^3-yr). The majority of pulmonary function parameters declined with exposure category: for example, after standardization for age, height, and race, FEV_1 declined from 3.80 L at < 50 mppcf-yrs to 2.94 L at > 400 mppcf-yrs. The corresponding values expressed as a percentage of the values obtained from internal referents with minimal asbestos exposure were 98% and 92%, respectively. Reductions were generally greater in those subjects with any asbestos-related radiographic changes, and in the lowest exposure groups, reductions were only seen in those with x-ray changes. There was a significant relationship between total dust exposure and most measures of lung function, including TLC, VC, FVC, FEV_1, and $FEF_{25\%-75\%}$, but not residual volume, diffusing capacity, RV/TLC, and FEV_1/VC. Smokers had reduced lung function as compared to non-smokers. However, after controlling for smoking in the analysis, the different smoking groups did not differ significantly with respect to the relationship between asbestos exposure and pulmonary function and it was concluded that asbestos effects were not influenced appreciably by smoking habits (Weill et al., 1975).

Workers aged 44-59 at the time of the cross-sectional study (Weill et al., 1975) were re-evaluated in 1973 and 1980, and out of 244 eligible men, 133 participated (Jones et al., 1989). Both pleural and parenchymal abnormalities had progressed at the time of follow-up, and dust exposure was a significant predictor of progression for both types of radiographic abnormalities. Smoking, initial radiographic status, and average dust exposure all significantly affected FVC, FEV_1, and $FEF_{25\%-75\%}$. When subjects were followed through to 1980, annual changes in lung function were modest, with a decline in FVC and FEV_1 of 17 mL/year and 20 mL/year, respectively, estimated from a multiple linear regression that controlled for smoking. Radiographic status and continuous dust exposure were not significantly related to longitudinal lung function changes. Smoking remained as a significant determinant of declines in spirometry and increases in lung volumes. Therefore, it appeared that the adverse effects of asbestos exposure on lung function occurred prior to the longitudinal study, since adverse effects were clearly shown in the 1970 cross-sectional survey (Weill et al., 1975).

In a larger series of studies, an age-stratified random sample of 1015 men were selected from 6180 male asbestos miners and millers employed in Quebec in 1966 (Becklake et al., 1972). Similarly to Weill et al. (1975), dust exposure was divided into six exposure groups, ranging from < 10 to > 800 mppcf-yrs. After controlling for age, height, and weight, TLC, FVC, and FEV_1 fell with increasing dust exposure index for both smokers and non-smokers. Flow rates such as FEV_1/FVC and maximal mid-expiratory flow (MMEF) were lower in smokers than non-smokers; these flow rates were reduced substantially in the higher dust-index groups, regardless of smoking status. The FEV_1 of non-smokers fell from 4.11 L at < 10 mppcf-yrs to 2.25 L at > 800 mppcf-yrs, while the corresponding values for smokers were 3.84 L and 2.26 L, respectively. The potential influence of asbestos-related radiographic changes was not addressed in this study.

In 1974, 445 of the original 1015 men were re-evaluated by questionnaire and lung function tests, and more than half of them had chest radiographs close to the time of follow-up (Becklake et al., 1982). Actual values for pulmonary function measures were not provided in this paper, only the proportion of subjects whose pulmonary function parameters changed by more than 20% at follow-up as compared to the baseline measures. Age and smoking were significantly associated with development and progression of MMEF and FVC abnormalities, but asbestos exposure at baseline was not. In the 277 subjects with chest radiographs, exposure appeared to be related to parenchymal change progression, after controlling for age and smoking. The overall conclusion was that lung function abnormalities measured in a cross-sectional manner were related to past exposure levels, but that further progression and development of lung function abnormalities did not vary between exposure groups (Becklake et al., 1982).

Further combined cross-sectional and longitudinal studies were performed on a smaller group of asbestos-cement production workers (Siracusa et al., 1984; Siracusa et al., 1988). Of seventy-seven asbestos workers who completed spirometry in 1973, 65 were reassessed in 1980, and 61 of these men also completed spirometry in 1984. Asbestos (predominantly chrysotile) in a proportion of 15-20% was blended with portland cement. No silica was added in the production process. In 1973, dust sampling in the asbestos-cement mixing area was performed. Although airborne asbestos levels were not reported, total dust levels in the cement mixing area ranged from 3.30 to 32.10 mg/m^3, with a geometric mean of 10.88 mg/m^3. Dust exposure was dichotomized based on job type: those working in the very dusty mixing and grinding areas were defined as high exposure, while workers in all other asbestos cement areas were defined as low exposure. The analysis controlled for age, height, and smoking, but potential radiographic changes from asbestos exposure were not assessed.

In the 1980 analysis, FVC and FEV$_1$ were both roughly 600 mL lower in the high exposure group as compared to the low exposure group. Equivalent values expressed as a percentage of predicted were 101% (FVC) and 97% (FEV$_1$) for subjects in the high exposure group versus 110% and 108%, respectively, for subjects in the low exposure group. Based on a multiple regression analysis, the annual decline in FVC and FEV$_1$ attributable to the number of years since first asbestos exposure was 16 mL per year. Cross-sectional estimated yearly changes in FVC and FEV$_1$ for 1973 and 1980 were very close to the actual observed decline (approximately 50 mL decline per year for both parameters). Subjects with less than 15 years of exposure had annual declines in FEV$_1$ and FVC of 38 mL and 24 mL, respectively, whereas those with more than 15 years exposure had annual declines of 51 mL and 53 mL. The combined effects of smoking and asbestos exposure were less than additive (Siracusa et al., 1984). In the 1984 survey, workers who were heavily exposed to asbestos had greater declines in FVC and FEV$_1$ than the low exposure group. Twenty years after first asbestos exposure, the excessive decline in FVC and FEV$_1$ for the heavily asbestos-exposed workers was estimated to be 500 mL more than workers who had low asbestos exposure (Siracusa et al., 1988).

In a 1976 cross-sectional survey, 125 asbestos cement workers with at least 10 years of employment were compared with 76 referent workers without asbestos exposure who had attended the same industrial health centre (Ohlson et al., 1984). Exposure levels in the asbestos cement plant were approximately 2 fibres per mL in the 1950's and 1960's, and 1 fibre per mL in the 1970's. After adjustment for age, height, tracheal area, and smoking, the FEV$_1$ and FVC of the asbestos-exposed subjects were 300 mL and 250 mL less, respectively, than the non-asbestos-exposed referents (Ohlson et al., 1984).

The plant had closed down at the time of the 1976 survey, and no further asbestos exposure occurred. Seventy-five asbestos cement workers and 56 referent workers from the original 1976 assessment were re-evaluated in 1980 (Ohlson et al., 1985). A different spirometer was used in the 1980 survey. Predicted spirometry values for asbestos-exposed subjects were calculated from the regression equations of the referents' 1980 data. For the 1980 data alone, there was an exposure-response relationship between fibre-years of asbestos exposure and percent predicted FVC and FEV_1; for workers with less than 14 fibre-years of exposure, FVC and FEV_1 were both close to 100% of the predicted values but for workers with greater than 23 fibre-years of exposure, FVC was 88% predicted and FEV_1 was 86% predicted. Longitudinally, four-year decrements in FVC and FEV_1 were larger for the asbestos-exposed subjects than the referents, and the decrements increased by fibre-year category. The loss of FVC and FEV_1 was 8.2% and 8.7% respectively, from 1976 values for those with greater than 23 years of exposure, as compared to 4.5% and 5.6% declines, respectively, for controls. After controlling for age, height, and tracheal area, FEV_1 was significantly related to cumulative asbestos exposure and smoking, but not to the presence of pleural plaques. It is noteworthy that for the non asbestos-exposed referents, the annual declines in FVC and FEV_1 were about 50 mL per year, well above the roughly 20 to 30 mL annual declines typically estimated from population surveys (ATS, 1991). The authors suggested that the spirometer used for the 1976 survey might have given artificially inflated values, as it was not properly calibrated. This would not have changed the comparison between asbestos workers and referents, but it would have invalidated the degree of the differences.

Recently, Alfonso et al., (2004) reported on the lung function of former workers and residents of Wittenoom, Australia, where crocidolite asbestos was mined from 1943 to 1966. Follow-up on cohorts of former workers and residents was initiated in 1979, and the study reported the results of spirometric measurements performed on 1392 subjects between 1992 and 2002. Crocidolite exposure for ex-workers was determined by multiplying results from airborne sampling performed at worksites in 1966 by the length of time in particular jobs, while exposure for residents relied on surveys of fibre concentrations in the township conducted periodically by the Health Department of Western Australia. Average cumulate crocidolite asbestos exposure for residents and workers were 6.9 and 24.7 fibres/mL-yr, respectively. Eighteen percent of workers and 1% of residents had radiographic asbestosis. On average, FEV_1 and in FVC declined by 24 mL and 39 mL per year, respectively. Each additional fibre/mL-year of asbestos exposure was associated with a decrease in FEV_1 of 0.9 mL and in FVC of 1.5 mL; cumulative exposure was not a significant predictor of FEV_1/FVC. The presence of asbestosis was associated with an additional decrease of 13 mL/year in FEV_1 and 20 mL per year in FVC. Current smokers had significantly lower FEV_1, FVC, and FEV_1/FVC than never smokers, but there were no significant interactions between asbestos exposure and smoking history on the level and rate of decline of lung function, suggesting that smoking and asbestos exposure acted independently.

A cohort of 828 Brazilian workers employed in the manufacture of asbestos-cement products were studied between 1995 and 1999 (Algranti et al., 2001). Using predicted values derived from a Brazilian population sample, mean predicted FEV_1 and FVC declined with increasing radiographic profusion grades. Percent-predicted FEV_1 and FVC declined significantly with increasing indices of exposure (years since first exposure, years of exposure, and cumulative exposure). Trends were similar for these two parameters for smokers and non-smokers, but the changes with dust exposure were greatest for smokers: mean percent-predicted FEV_1 and FVC were each 7% lower in the highest exposure quartile as compared to the lowest exposure quartile for non-smokers, while for smokers, the corresponding difference was in the range of 13% to 16%. For FEV_1/FVC, the only significant exposure index was years since first exposure. FEV_1/FVC did not decline with increasing quartiles of exposure indices for non-smokers. Spirometry was normal in 71% of subjects, 19% had an obstructive defect (FVC normal and FEV_1/FVC below lower 95% confidence limit from the predicted values), and 8% had a restrictive defect (FEV_1/FVC normal and FVC below lower 95% confidence limit from the predicted values). Using logistic models, having an obstructive defect was significantly associated with smoking, age, and BMI, and having a restrictive defect was significantly associated with asbestosis and years since first asbestos exposure.

Erdinc et al. (2003) studied the effects of chrysotile asbestos exposure in 74 Turkish brake-lining production workers and 12 unexposed office workers using chest radiographs and pulmonary function tests from assessments in 1992 and 1999. None of the exposed workers had radiographic asbestosis. The twenty-five non-smoking asbestos workers had significantly lower percent-predicted $FEF_{25\%}$ and diffusing capacity in 1999 as compared to 1992. For the 49 smoking asbestos workers, the above two parameters plus FEV_1 and FVC were significantly lower in 1999.

2.3.3 Studies of Workers in Specific Trades

Specific trades at high risk for asbestos exposure include insulators, sheet metal workers, plumbers and pipefitters, steamfitters, boilermakers, and numerous shipboard trades (Brodkin et al., 2005). Several large studies have been performed with subjects recruited though trade unions.

One of the largest studies of heavily exposed asbestos workers was conducted by Miller et al. (1992). This cohort of 2611 asbestos insulators was evaluated in 19 cities in the United States and Canada between 1981 and 1983. Close to 87% of subjects were evaluated at least 30 years from the onset of asbestos exposure. Sixty percent of subjects had chest radiographs with ILO scores of 1/0 or greater, indicating asbestosis. Predicted spirometry values were derived from a random population sample from the state of Michigan. In non-smokers, FEV_1 and FVC were reduced (87% predicted and 85% predicted, respectively) but measures of airflow, such as FEV_1/FVC, $FEF_{25\%-75\%}$, and $FEF_{75\%}$ were close to 100% of predicted. Values were lower in smokers, with FEV_1 75% of predicted, FVC 82% of predicted, and measures of airflow 6-24% lower than in the non-smokers. There was a highly significant inverse relationship between FVC and ILO profusion score, for both smokers and non-smokers. Workers without radiographic asbestosis (ILO = 0/0) also had a reduced FVC at 88% predicted. FVC was lower in subjects with pleural thickening (diffuse and circumscribed) as compared to those without at equivalent profusion scores; for example in subjects with ILO profusion 1/1 and 0/0, FVC was 8% lower and 3.6% lower, respectively in subjects with pleural thickening versus those without pleural thickening. Diffuse pleural thickening was associated with a greater FVC decrement than circumscribed thickening at every profusion score (Miller et al., 1992).

The same subjects were re-analyzed in a subsequent paper that focused on pulmonary impairment categories (Miller et al., 1994). The frequency of subjects with restrictive impairment (FVC less than the lower 95% confidence limit of predicted value and normal FEV_1/FVC) and combined restrictive and obstructive impairments (FVC less than the lower 95% confidence limit of predicted value and FEV_1/FVC below 0.70) increased with increasing duration from onset of exposure. A regression analysis used the independent variables of smoking pack years, radiographic category, and duration from onset of exposure. For FVC, each year from the onset of asbestos exposure and each pack year decreased FVC by 0.44% and 0.096% of predicted, respectively. For a duration from exposure onset of 35 years, FVC decreased by 15.3%, whereas for an average smoking history of 41 pack years, FVC decreased 4.0%. Radiographic pleural involvement and pleural involvement plus parenchymal involvement decreased FVC by 4.8% and 7.5% of predicted, respectively. For FEV_1/FVC, the influences were smoking (ratio decrease of 4.6% for 41-pack-year history), duration from onset of exposure (ratio decrease of 3.5% for 35 years since initial exposure), and parenchymal disease on chest radiograph (1.2% decrease in FEV_1/FVC ratio). When the analysis was restricted to non-smokers only (515 subjects), results for FVC were similar to the entire cohort, whereas for FEV_1/FVC, only radiographic parenchymal involvement had any significant effect. The predominant influence on a restrictive impairment was years since initial asbestos exposure, and this effect was slightly greater in smokers. There was little evidence that asbestos exposure alone lead to an obstructive impairment, as duration from onset of exposure did not predict FEV_1/FVC for non-smokers, and only 6% (31/515) of non-smokers had an obstructive impairment. It was also noteworthy that in this population, 27% of subjects with a normal chest radiograph had either restrictive or combined restrictive/obstructive impairment.

Further to their study of asbestos insulators (Miller et al., 1994), 1295 sheet metal workers with lower intensity asbestos exposure than the insulators were assessed in a cross-sectional study (Miller et al., 1996). Workers were surveyed through their unions and all had at least 20 years in the trade. Similarly to the insulators, FVC fell significantly with increasing ILO profusion score in sheet metal workers. The slope of the decrease was significantly lower in the sheet metal workers as compared to the insulators. At each profusion score, FVC was consistently lower in the presence of pleural thickening. Even workers with radiographically normal lung fields (ILO grade 0/- or 0/0) had decreased FVC values (88% predicted of reference values derived from a random population sample from an industrial state). As well, age-adjusted FVC values decreased with increasing duration of exposure in those with normal lung fields. Based on a multiple regression analysis, each increment in ILO grade was associated with a decrease in FVC of 0.55% predicted in the sheet metal workers.

Glencross et al. (1997) conducted a longitudinal study of sheet metal workers. Of 331 workers who attended a voluntary union screening program in 1981-1982, 122 attended a follow-up evaluation in 1991. Seventy-four percent of the workers had no impairment on spirometry, using cut-offs of less than 80% of predicted values. In a multivariate regression model, age, height, smoking, and previous shipyard work were significant predictors of annual decline in FVC; year of initial asbestos exposure, total years of asbestos exposure, and pleural plaques were not related to FVC decline. Among smokers, age and a history of ship work were significant predictors of larger annual losses in FEV_1. Although this study demonstrated that in smokers, asbestos exposure from shipyard work led to accelerated losses in FVC and FEV_1, the findings are limited due to the losses of subjects to follow-up and lack of control for other non-asbestos exposures such as welding, painting, metal grinding, and sand blasting.

Another large cross-sectional study examined North American active and retired union members (boilermakers, iron ship makers, plumbers, pipefitters, and others), and included 1146 subjects with asbestosis (ILO profusion grade 1/0 or greater) and 1146 subjects with asbestos exposure but no asbestosis (Kilburn et al., 1994). A further group of 370 men without asbestos exposure who were derived from a population sample were also included in the study for comparison purposes. The 119 asbestos-exposed non-asbestosis workers who were never smokers had significantly lower $FEF_{75\%-85\%}$ and FEV_1/FVC and higher TLC as compared to the non asbestos-exposed non-smoking referents. Men with asbestosis were not significantly different than the asbestos-exposed men without asbestosis on any of the pulmonary function measures, but were significantly different than the non-exposed referents on all measures. Similar results were found for smokers when comparing asbestos-exposed workers without asbestosis to the smoking referents, except there were also significant differences for FEV_1 and $FEF_{25\%-75\%}$. Smokers with asbestosis were significantly different from referents on all measures, and also had significantly lower FVC and $FEF_{75\%-85\%}$ and higher TLC as compared to asbestos-exposed workers without asbestosis. In a regression analysis, as the radiographic profusion of opacities increased from 0/0 to 3/2, FVC, FEV_1, FEV_1/FVC, $FEF_{25\%-75\%}$, and $FEF_{75\%-85\%}$ decreased in non-smokers. Increases in radiographic asbestosis severity corresponded to greater airways obstruction, as measured by $FEF_{25\%-75\%}$. Similar relationships were found in smokers, except that duration of asbestos exposure explained more of the variance than ILO profusion score in the non-smokers whereas the reverse relationship was true in the smokers. The overall conclusion was that asbestos exposure caused airway obstruction and that this impairment worsened more rapidly in smokers. As opacities become visible on chest x-ray as an indication of asbestosis, mid-expiratory flows, FEV_1, and FVC decreased significantly, but TLC increased due to air trapping.

Two small studies examined different asbestos-exposed tradespeople in Alberta. In one study, Hessel et al. (1998a) compared 102 actively employed boilermakers with 100 telephone workers who had minimal dust exposure. All workers had at least 20 years of union membership. Standard radiographs revealed minimal asbestos-related changes. There was no difference in lung function parameters between the boilermakers and telephone workers. However, when the boilermakers were divided into those who worked primarily as boilermakers (n=50) and those who had their longest service as a welder (n=52), the primary boilermakers had significantly lower percent-predicted FEV_1 (96% vs. 103%), FEV_1/FVC (94% vs. 98%), MMEF (84% vs. 99%), and FEF_{50} (86% vs. 101%). These differences were attributed to greater dust exposure in the primary boilermakers. The other study compared 99 actively employed plumbers and pipefitters, all with at least 20 years of union membership, with telephone workers (Hessel et al, 1998b). Bilateral pleural changes (including plaques and diffuse thickening) was observed in 7.8% of plumbers and pipefitters and 1% of telephone workers. There was no radiographic evidence of interstitial disease in both either subject group. The only significant pulmonary function difference between the two groups of workers was a lower FVC among never and ex-smoker plumbers and pipefitters, but when workers with any radiographic abnormalities were excluded, none of the differences between plumbers and pipefitters and telephone workers were significant.

2.3.4 Influence of Radiographic Pneumoconiosis and Pleural Changes

Several of the previously mentioned studies have consistently shown that pulmonary function decreases in a linear fashion with increasing severity of radiographic asbestosis, as measured by ILO profusion score (Algranti et al., 2001; Kilburn et al., 1994; Miller et al., 1992; Miller et al., 1996). It has even been suggested that the effect of asbestos exposure alone, in the absence of asbestosis, does not have a significant effect on FEV_1 or FVC (Nakadate, 1995).

However, pulmonary function decrements have also been observed in subjects without radiographic asbestosis (Erdinc et al., 2003; Kilburn et al., 1994; Miller et al., 1992; Miller et al., 1996). In their study of 681 plumbers and pipefitters, Rosenstock et al. (1988) demonstrated that both FVC and FEV_1 declined in a linear fashion with increasing ILO profusion score, regardless of smoking status. They also observed that there was a significant difference in percent-predicted FVC between ILO grades 0/0 and 0/1, as well as grades 0/1 and 1/1, indicating that pulmonary function decrements could exist in the absence of radiographic asbestosis (ILO grade 1/0 and greater).

The observation that lung function can be affected in the absence of radiographic pneumoconiosis visible on a chest radiograph is consistent with other studies of dust-exposed workers, and supports the poor sensitivity of this imaging modality in detecting early parenchymal fibrotic changes, as compared to more sensitive imaging techniques, such as high-resolution computed tomography (HRCT) (Neri et al., 1996; Lebedova et al., 2003; Soulat et al., 1999). Neri et al. (1996) used HRCT to evaluate 119 asbestos-exposed shipyard workers who had no clinical symptoms of lung disease. Although none of the workers had evidence of asbestosis on plain radiographs (all had an ILO profusion score less than or equal to 0/1), 7 had parenchymal abnormalities alone, and 31 had both pleural and parenchymal involvement. There were no significant differences in pulmonary function parameters between workers with pleural lesions and those without. However, those with parenchymal lesions had significantly lower mean FEV_1 than those with normal parenchyma (86% of predicted versus 92% of predicted). When the subjects were analyzed separately based on smoking habit, the 10 non-smokers with parenchymal lesions had significantly lower FVC than the 34 non-smokers with normal parenchyma. Among present and ex-smokers, the only significant pulmonary function parameter difference was a lower mean FEV_1/FVC in those with parenchymal abnormalities compared to those without.

The greater sensitivity of HRCT was also demonstrated by Soulat et al. (1999), who studied 170 ex-factory workers who had intermittent exposure to asbestos during maintenance operations. Only 9 subjects had visible parenchymal abnormalities on standard chest radiographs, but 34 subjects had parenchymal abnormalities visible on HRCT. Unlike the results of Neri et al. (1996), in this study there were no significant differences in FEV_1, FVC, MEF (maximal expiratory flow), or MMEF (maximal mid-expiratory flow) between subjects with and without pleural thickening or parenchymal densities on HRCT scan. The authors concluded that the presence of pleural plaques or mild parenchymal fibrosis were not associated with significant pulmonary function impairment.

The discrepancies in the relationship between parenchymal fibrosis visible on HRCT and pulmonary function in the Neri et al. (1996) and Soulat et al., (1999) might be explained by other factors, such as the presence of emphysema. Piirilä et al. (2005) studied 590 asbestos-exposed subjects (only 3% were never smokers) and used HRCT to evaluate both fibrosis and emphysema. The subjects in the study had only slight or moderate lung fibrosis. FEV_1 and FVC impairment were graded based on comparison to a reference population as follows: ≥81% = normal, 65-80% = slight, 45-64% = moderate, and ≤44% = severe. Compared to the normal category, slight, moderate, or severe impairment of FEV_1 was positively associated with combined fibrosis and emphysema. Emphysema scoring assessed with HRCT appeared to be a strong determinant of lung function impairment in subjects with both emphysema and fibrosis, who had the most severely reduced FEV_1, DLCO, and TLC values. Those with lung fibrosis alone demonstrated only slight ventilatory impairment, even though their fibrosis score approached that of persons with combined disease. Piirilä et al. (2005) concluded that emphysema in asbestos-exposed workers was the predominant factor in impairment of pulmonary function in persons with slight to moderate asbestosis and that smoking appeared to be most important cofactor in development of functional impairment.

In addition to parenchymal abnormalities associated with asbestos exposure, asbestos-induced pleural changes can also influence pulmonary function. These pleural changes are broadly grouped into circumscribed pleural thickening (pleural plaques), and diffuse pleural thickening (pleural fibrosis), the latter being more extensive. Results from studies that have examined the effects of pleural plaques have been somewhat mixed. Some of the previously mentioned studies suggest that PFT changes are not related to the presence of pleural plaques (Glencross et al., 1997; Neri et al., 1996; Ohlson et al., 1985). This conclusion is also supported by a recent HRCT study of 73 male asbestos-cement factory workers and 21 non asbestos-exposed referents (Cleemput et al. 2001). None of the exposed workers had radiographic asbestosis but localized pleural plaques were detected in fifty-one (70%) of the workers. There was no significant difference in lung function parameters (FVC, FEV_1, FEV_1/FVC, $FEF_{25\%}$, $FEF_{50\%}$, $FEF_{75\%}$, peak expiratory flow, and diffusing capacity) between workers with plaques and workers without plaques, nor with non asbestos-exposed referents.

Conversely, several other studies have observed a relationship between pleural plaques and pulmonary function decrements. Oliver et al. (1988) examined 359 male railroad workers who had chest radiograph profusion of less than 0/1 and no diffuse pleural thickening. Almost one quarter of the subjects had pleural plaques. The men with plaques had significantly lower absolute and percent-predicted values for FVC and FEV_1. After controlling for smoking and duration of employment, pleural plaques were significantly associated with percent-predicted FVC, but not FEV_1. The presence of pleural plaques was associated with a 4.3% decrement in FVC and a 2.1% decrement in FEV_1. It was concluded that the presence of asbestos-related pleural plaques plus a restrictive ventilatory defect could indicate occult interstitial fibrosis. Kilburn et al. (1990) studied 79 men with diaphragmatic pleural plaques as the only radiographic sign of asbestos disease. Asbestos-exposed smokers had significantly reduced percentage predicted expiratory flows (FEV_1, $FEF_{25\%-75\%}$, $FEF_{75\%-85\%}$, and FEV_1/FVC), compared with referent smokers, after adjusting for duration of smoking. The 21 asbestos-exposed non-smokers had significantly lower FEV_1, FEV_1/FVC, and $FEF_{75\%-85\%}$ as compared to referents. For both smokers and non-smokers, there was no difference in FVC between the asbestos-exposed workers and referents. In this group of asbestos-exposed workers with diaphragmatic plaques but no radiographic evidence of asbestosis, decreased expiratory flows and air trapping was the primary physiologic lesion, without any evidence for a restrictive impairment.

Schwartz et al. (1990) studied 1211 active and retired members of a sheet metal workers' union through a nationwide medical evaluation in the United States. Subjects had at least 25 years in the trade, and 17% had an ILO profusion score of 1/0 or greater. Subjects were grouped broadly into "normal pleura", "circumscribed plaques", and "diffuse pleural thickening". Workers with diffuse pleural thickening had the most extensive interstitial fibrosis, were the oldest, had the most years in the trade, and had the lowest proportion of never smokers; the opposite was true for workers with normal pleura, and workers with circumscribed plaques were in the middle. Multivariate analyses that included age, height, pleural fibrosis, ILO profusion category, years since first employed in the sheet metal trade, and smoking revealed that both pleural plaques and diffuse pleural thickening were associated with declines in FEV_1 and FVC, but not FEV_1/FVC, indicating a restrictive pattern of impairment. As compared to workers with normal pleura, the mean decline in FVC was 270 mL for those with diffuse pleural thickening and 140 mL for those with circumscribed plaques. The corresponding FVC declines for these two groups of subjects for ILO category 1 profusion was 280 mL and 280 mL, respectively, and for category 2 profusion were 1020 mL and 870 mL, respectively. A decline in FVC of 10 mL per year since first employed in the sheet metal trade was observed for both pleural thickening groups. Comparison of percent predicted FVC across pleural thickening categories and stratified by presence or absence of interstitial fibrosis illustrated the cumulative effect of interstitial fibrosis and pleural fibrosis. For workers without interstitial fibrosis, the percent-predicted FVC for no pleural fibrosis, circumscribed plaques, and diffuse thickening were 95%, 90%, and 86%, respectively, while the corresponding values for workers with interstitial fibrosis were 83%, 80%, and 74%.

A more recent HRCT study of 162 asbestos-exposed workers also supported the findings of Schwartz et al. (1990). Pleural lesions were categorized based on number, width, and thickness, and parenchymal changes were categorized as asbestosis or suspected asbestosis. Parenchymal abnormalities were significantly more frequent in subjects with pleural lesions than those without (67% vs. 15%). After adjustment for the effects of parenchymal fibrosis, subjects with pleural lesions had significantly lower FEV_1 and FVC than subjects without pleural lesions, but this was only true for higher categories of pleural lesions (Lebedova et al., 2003).

As noted in the studies of Schwartz et al. (1990) and Lebedova et al. (2003), pleural plaques are frequently a marker of asbestos dose, and subjects with pleural changes are also more likely to have parenchymal changes consistent with asbestosis. Disentangling the effects of pleural plaques independent of parenchymal fibrosis is challenging, and the crude methods used to control for asbestosis in the above studies may not be adequate to fully explore these relationships. Although there is conflicting evidence as to whether or not the presence of pleural plaques alone affects lung function, there is consistent evidence that diffuse pleural thickening (pleural fibrosis) negatively impacts lung function, independent of asbestosis (Miller et al., 1992; Miller et al., 1996; Rosenstock et al., 1988).

The independent effects of diffuse pleural thickening were evaluated in a study of 106 subjects from the same exposure group, 53 with diffuse pleural thickening and 53 without (Kee at al., 1996). There was no significant difference between the two groups with regards to age, smoking history, duration of asbestos exposure, latency from initial exposure, pleural plaques, or interstitial fibrosis. Those with diffuse pleural thickening had significantly lower FVC and diffusing capacity than those without pleural thickening. Multivariate analyses revealed that subjects with either diffuse pleural thickening alone or diffuse pleural thickening with interstitial fibrosis had significantly lower FVC and diffusing capacity than subjects with interstitial fibrosis alone.

Lilis et al. (1991) used a radiographic index score to examine the effect of the extent of pleural fibrosis on pulmonary function in 1584 asbestos insulation workers who were a subset of the study population of Miller et al. (1992). Seventy-five percent of the subjects had either circumscribed thickening or diffuse pleural fibrosis. A gradual decrease in percent predicted FVC was observed with increasing pleural fibrosis index, and this was most marked in subjects who also had parenchymal fibrosis. Circumscribed pleural plaques had a much lower quantitative effect on FVC than diffuse pleural fibrosis, such that the highest index score for circumscribed pleural fibrosis was associated with a higher FVC than the lowest index score for diffuse pleural fibrosis. The initial impact of diffuse pleural thickening on pulmonary function was confirmed in a longitudinal study of 36 subjects with diffuse pleural thickening who were followed over 9 years (Yates et al., 1996). There was no correlation between changes in radiographic score and longitudinal changes in FEV_1 or FVC, leading the authors to conclude that diffuse pleural thickening was associated with an initial loss of lung function, followed by relative stability in most cases.

2.3.5 Influence of Smoking

In the studies discussed above that have examined the effects of smoking and asbestos exposure simultaneously, it has been consistently observed that the adverse pulmonary function effects associated with asbestos exposure are greater in smokers as compared to non-smokers (Alfonso et al., 2004; Algranti et al., 2001; Erdinc et al., 2003; Kilburn et al., 1994; Miller et al., 1992; Ohlson et al., 1984; Siracusa et al., 1984; Weill et al., 1975). The combined effects of smoking and asbestos exposure have been described as additive (Alfonso et al., 2004) and less than additive (Siracusa et al., 1984).

It has also been commonly observed that, although of smaller magnitude as compared to smokers, declines in pulmonary function are also observed in non-smokers, especially if radiographic asbestosis is present (Algranti et al., 2001; Becklake et al., 1972; Erdinc et al., 2003; Kilburn et al., 1994; Miller et al., 1994; Nakadate, 1995; Siracusa et al., 1984; Weill et al., 1975). Additional evidence for the deleterious effects of asbestos exposure in the absence of smoking come from studies that have specifically studied only non-smokers, several of which are summarized below.

In a study of 331 Quebec asbestos miners and millers who had been referred for evaluation to the local Workmen's Compensation Committee, non-smokers with asbestosis had significantly lower diffusion capacity, lung volumes, and airflow conductance as compared to manual workers without asbestos exposure (Begin et al., 1987). The non-smoking asbestos-exposed workers only differed from the manual workers by having a 30% decrease in upstream airflow conductance, which would cause mild airflow limitation at low lung volumes, but would not significantly reduce expiratory flow rates. In a smaller study of non-smoking Quebec miners and millers, 7 subjects with asbestosis and 10 without asbestosis were compared with 16 age and height-matched lifetime non-smoker control subjects (Begin et al., 1983). The workers with asbestosis had significantly lower TLC, VC, and carbon monoxide diffusing capacity as compared to the non-asbestosis workers. For the most part, the asbestos-exposed workers without asbestosis were no different than unexposed controls. However, flow rates at low lung volumes were somewhat lower than controls in these workers, suggesting minimal peripheral airway obstruction.

Rom (1992) studied a select group of men with asbestosis (radiographic opacities graded as 1/0 or greater) who were either lifelong non-smokers or who were ex-smokers of greater than 5 years. Seventy-seven subjects were evaluated more than once from 1983 to 1989. Radiographic opacities and rales on physical examination were significantly associated with longitudinal decrements in VC, FEV_1, and TLC, consistent with an asbestosis-related restrictive impairment. Overall, the 77 subjects had higher annual declines in lung function parameters than would be expected in a normal population. For example, FVC and FEV_1 declined 92mL/year and 66 mL/year, respectively.

Twenty-three asymptomatic non-smoking plumbers with rare exposures to welding fume, cement dust, or irritant gases were compared with 23 non-smoking electricians (Dossing et al., 1990). The two groups of workers were similar with regards to age, height, weight, years of employment, and participation in sporting activities. Total lung capacity and maximal expiratory flow at 50% and 25% of FVC were significantly lower in the asbestos-exposed plumbers. Based on the results of ventilation scintograms, 11 of the 23 plumbers had findings that were consistent with airways obstruction. In a small study of 45 non-smoking shipyard workers, 13 subjects had maximal mid-expiratory flow rates that were less than 75% of predicted, suggestive of obstruction of small airways (Mohsenifar et al., 1986).

Further support for the effects of asbestos on small airways in the absence of smoking was provided by a cross-sectional study of 416 asbestos insulators, 97 of who were non-smokers (Kilburn et al., 1985). As compared to a Michigan population sample and adjusted for age and height, non-smoking insulators had significantly lower terminal airflows, as measured by $FEF_{75\%-85}$, and FEV_1. FVC was 124 mL lower in the non-smoking insulators, but this was not statistically significant. The authors concluded that in the absence of smoking, asbestos exposure lead to decreased airflow in small airways and stiffening of the lung parenchyma.

Wang et al. (1998) compared 208 female non-smoking asbestos textile and shingle factory workers without radiographic evidence of asbestosis with 136 female non-smoking control workers from a nearby electronic instrument manufacturing plant. There were no substantial dust exposures in the factory other than from asbestos. Compared to controls, height-adjusted parameters that were significantly lower in the asbestos workers were diffusing capacity, VC, FVC, and $FEF_{25\%-75\%}$. When the workers and controls were each split into 2 groups based on age (younger than 35 years of age and older than 35 years of age), the only parameter that was significantly different between the younger asbestos workers and corresponding controls was VC. The older asbestos workers (mean age 42 and average exposure duration 16 years) had significantly lower VC, FVC, and $FEF_{25\%-75\%}$ as compared to the older controls. Estimated annual losses for FVC were 8.7 and 2.0 mL per year for the younger asbestos workers and controls, respectively, but 41.7 and 10.6 mL per year for the older asbestos workers and controls, respectively. The authors concluded that even in the absence of radiographic asbestosis and effects of smoking, exposure to asbestos is associated with pulmonary function loss, predominantly via lower lung volumes.

In a subsequent study by the same research group, 269 male and female workers from the same asbestos textile and shingle factory were compared with 274 male and female workers without industrial dust exposure from a nearby factory (Wang et al., 2001). All of the female workers in the study were non-smokers. One third of the exposed males had radiographic asbestosis, one fifth had pleural thickening, and 6% had pleural plaques; 17% of the exposed females had radiographic asbestosis, and 5% each had pleural thickening and pleural plaques. As a group, asbestos-exposed workers had significantly lower TLC, FVC, FEV_1, $FEF_{50\%}$, $FEF_{25\%}$, diffusing capacity, and RV/TLC ratio. When the males were categorized by smoking status, non-smoking asbestos workers had significantly lower TLC, FVC, and diffusing capacity compared to the non-exposed referents. Measures of airflow (FEV_1, $FEF_{50\%}$, and $FEF_{25\%}$) were lower but not significantly. Linear regression analysis demonstrated that asbestos exposure was associated with lower TLC, FVC, FEV_1, $FEF_{50\%}$, $FEF_{25\%}$, and diffusing capacity. This study confirmed the findings of their previous report in that amongst non-smokers, asbestos exposure was associated with restrictive defects and airflow limitation, especially at lower lung volumes.

2.3.6 Asbestos Summary

Several studies have consistently observed a dose-response relationship between inhalation exposure to asbestos fibres and pulmonary function impairment (Alfonso et al., 2004; Algranti et al., 2001; Becklake et al., 1972; Finkelstein et al., 1986; Weill et al., 1975). Affected pulmonary function indices have included diffusing capacity, TLC, FVC, FEV_1, FEV_1/FVC, MMEF, and other markers of airflow at sub maximal lung volumes, indicative of both restrictive and obstructive defects.

Estimates of the absolute negative effects of asbestos exposure on pulmonary function have varied across studies, which is not unexpected given differences in exposure levels and study designs. The absolute FEV_1 and FVC values for subjects with high asbestos exposure have been observed to be 250 to 300 mL (Ohlson et al., 1985), 500 to 600 mL (Siracusa et al., 1988) and well over 1000 mL (Becklake et al., 1972) lower than subjects with low or no asbestos exposure. Miller et al. (1994) estimated that 35 years of asbestos exposure resulted in a decline in percent predicted FVC of 15%. Annual declines in FEV_1 and FVC for subjects with more than 15 years of asbestos exposure have been estimated to be just over 50 mL per year (Siracusa et al., 1988). Each additional fibre/mL-year of cumulative exposure has been associated with a decrease in FEV_1 of 0.9 mL and in FVC of 1.5 mL (Alfonso et al., 2004).

Decrements in pulmonary function are typically greater in the presence of radiographic asbestosis and also in smokers. Several of the previously mentioned studies have consistently shown that pulmonary function decreases in a linear fashion with increasing severity of radiographic asbestosis, as measured by ILO profusion score (Algranti et al., 2001; Kilburn et al., 1994; Miller et al., 1992; Miller et al., 1996). Although typically of lower magnitude, decrements in pulmonary function have been frequently observed in the absence of asbestosis on standard chest radiographs (Erdinc et al., 2003; Kilburn et al., 1994; Miller et al., 1992; Miller et al., 1996; Rosenstock et al., 1988). This likely reflects the low sensitivity of standard chest radiographs in detecting asbestosis, as HRCT often identifies fibrosis in subjects with normal chest radiographs (Lebedova et al., 2003; Neri et al., 1996; Soulat et al., 1999). The negative influence of asbestos-related pleural changes on pulmonary function appear to be independent of the degree of parenchymal fibrosis, but the evidence for this is more convincing for diffuse pleural thickening than for pleural plaques.

It has been consistently observed that the adverse pulmonary function effects associated with asbestos exposure are greater in smokers as compared to non-smokers (Alfonso et al., 2004; Algranti et al., 2001; Erdinc et al., 2003; Kilburn et al., 1994; Miller et al., 1992; Ohlson et al., 1984; Siracusa et al., 1984; Weill et al., 1975). Although generally of smaller magnitude, pulmonary function decrements in non-smokers have been consistently reported (Algranti et al., 2001; Becklake et al., 1972; Dossing et al., 1990; Erdinc et al., 2003; Kilburn et al., 1985; Kilburn et al., 1994; Miller et al., 1994; Mohsenifar et al., 1986; Nakadate, 1995; Rom, 1992; Siracusa et al., 1984; Wang et al., 1998; Wang et al., 2001; Weill et al., 1975).

3.0 Methods

3.1 Fibrosis Program Database Content

All information collected from individual workers under the Fibrosis program (x-ray plates, PFT spirograms, assessment forms, and all other collected information) was required to be forwarded to the main Alberta government office responsible for Program administration. Starting in the mid-1980's, Fibrosis Program information was coded and transferred into four computer files, with information for each specific worker linked across files by a 6-digit identification number that was unique for each worker. Data contained within the four computer data files were as follows:

a) The "Master" file contained basic demographic information for each worker, including name, birth date, sex, and province of birth, plus symptoms (cough, wheeze, colds, dyspnea, sputum, chest tightness, chest pain, fatigue), past illnesses (heart trouble, bronchitis, pneumonia, pleurisy, tuberculosis, asthma, hay fever, emphysema, other chest trouble), and occupational history (previous exposure to coal, quarry, foundry, asbestos, silica, or other dusty job, and years of employment in past jobs).
b) The "Dispdata" file contained multiple data entries for each worker, presumably corresponding to each examination that the worker attended. Each entry contained the examination date, an industry code, company identification number, exposure code (coal, silica, asbestos, other dust, man made mineral fibre), and smoking information (number of years of smoking, number of years since quitting smoking, years of cigar smoking, ex-smoker (yes or no), present smoker (yes or no)).
c) The "PFTdata" file contained multiple data entries for each worker, presumably corresponding to each examination that the worker attended. Each entry contained the examination date, the observed FEV_1 value, and the observed FVC value, expressed in litres. Height information was also contained within this file, expressed in centimetres, and typically a height value would be present only at the first of multiple examinations for a given worker.
d) The "Xraydata" file contained multiple data entries for each worker, presumably corresponding to each examination that the worker attended. Each entry contained the examination date, and an x-ray ILO code (0/-, 0/0, 0/1, 1/0, 1/1, 1/2, 2/1, 2/2, 2/3, 3/2, or 3/3).

For the above data, birth date, occupational history, years of smoking, height, observed FEV_1, and observed FVC were continuous variables and the remainder were categorical variables. Analyses were performed using data contained within these four data files.

3.2 Dependent Variables

3.2.1 FEV_1, FVC, FEV_1/FVC%

The dependent variables of interest were the observed FEV_1 and observed FVC values, expressed in litres in the "PFTdata" file. For subjects with at least one PFT examination, the earliest recorded FEV_1 and FVC values for each subject was defined as the first PFT. For subjects with at least two PFT data entries, the determination of the last PFT is described in Section 3.4. The FEV_1/FVC ratio was calculated by dividing the observed FEV_1 by the observed FVC; the result was then multiplied by 100 so as to be expressed as a percent value, FEV_1/FVC%.

3.2.2 Percent-Predicted FEV_1 and FVC

The Fibrosis Program was only intended to collect data on dust-exposed workers. The potential effects of dust exposure on pulmonary function in general could not be directly assessed through the use of a non-exposed referent group of workers, as this data was not collected as part of the Fibrosis Program.

In order to compare the Fibrosis Program workers with a non-exposed population, prediction equations derived from the general population were used to generate percent-predicted FEV_1 and FVC values. Although percent-predicted values were included within the "PFTdata" file, there was no record specifying which prediction values were used at varying times throughout the Fibrosis Program (Alleyne, 1982). As well, it was not until 1981 that the reference values of Morris et al. (1971) were identified as the specific equations to be used for determining predicted values (Fibrosis Program Information Manual, 1981).

Therefore, in order to ensure consistency, the prediction equations of Morris et al. (1971) were used to calculate percent-predicted values for all workers. According to the Fibrosis Program Information Manual (1981), there was some bias inherent in using Morris et al. norms as they were based on a sample of an American population that was not representative of the working Alberta population. However, they were recommended for use in the Fibrosis Program as they were based on a normal non-smoking healthy population, and they were one of the set of normal values recommended by the Task Force on Occupational Respiratory Disease (Fibrosis Program Information Manual, 1981).

For each worker who had adequate information on age, sex, and height, the predicted FEV_1 and FVC values were calculated as follows:

a) Predicted FEV_1 (male) =
$$(\text{height(cm)} * 0.3937 * 0.092) - (0.032 * \text{age}) - 1.260$$

b) Predicted FEV_1 (female) =
$$(\text{height(cm)} * 0.3937 * 0.089) - (0.025 * \text{age}) - 1.932$$

c) Predicted FVC (male) =
$$(\text{height(cm)} * 0.3937 * 0.148) - (0.025 * \text{age}) - 4.241$$

d) Predicted FVC (female) =
$$(\text{height(cm)} * 0.3937 * 0.115) - (0.024 * \text{age}) - 2.852$$

The observed FEV_1 and FVC values for each subject were then divided by the predicted values and multiplied by 100 in order to express the observed values as a percent of predicted.

3.3 Independent Variables

3.3.1 Date values

For ease of date calculations (eg. subtraction), all date entries were converted into numerical values consisting of four digits and two decimal places. The four digits represented the year and digits to the right of the decimal point represented the month and day as a proportion of a year. For example, following conversion, 1982.31 would correspond to April 22, 1982.

3.3.2 Exposure Type

At each examination, workers in the Fibrosis Program were assigned a one-digit exposure code, corresponding to coal, silica, asbestos, other dust, or man made mineral fibre (MMMF). Therefore, dust exposure in this analysis was simply a categorical value, and no information regarding the amount of exposure was available from the Fibrosis Program database.

During the analysis, it became apparent that two of the exposure categories, “silica” and “other dust”, might not have been mutually exclusive. When the broader subject pool was analyzed, it appeared as if “silica” became the preferred exposure code after 1982 for subjects who had previously been classified as “other dust” exposed. This is demonstrated in Figure 1, which depicts the frequency of “silica” and “other dust” exposure codes for subjects’ first PFT for each year of the Fibrosis Program. It can be observed that there is a substantial drop-off in the use of “other dust” as an exposure code at the same time that there is an increase in the use of “silica” as an exposure code. It was also observed that in subjects with multiple PFTs who were coded as “other dust” prior to 1982, their exposure code changed to “silica” after 1982, usually without a change in industry code or company identification number. Therefore, it was suspected that following the introduction of the Silica Regulation in 1982 (Alberta Regulation 9/82), silica-exposed workers who were originally classified as simply “dust-exposed” were subsequently coded as “silica-exposed”. However, this suspicion could not be confirmed. Therefore PFT entries with exposure codes of "silica" and "other dust" were combined into a single exposure group: "silica/other dust".

3.3.3 Duration of Exposure

Section 3.5 details the steps involved in the main analyses. The exposure durations of interest were dust exposure that occurred prior to the first Fibrosis Program PFT, and dust exposure that occurred prior to the last Fibrosis Program PFT for the subset of subjects with at least two PFTs. Exposure duration was expressed as a continuous variable.

In order to determine the duration of dust exposure prior to the first PFT, several data sources were examined:

> i) Firstly, an occupational exposure history (dust type and duration of exposure) for each subject was available from the "Master" file for almost all subjects with PFT outcome variables.
>
> ii) Secondly, for some subjects, their first "Dispdate" (presumably indicative of the date of entry into the Fibrosis program) did not correspond with the date of their first recorded PFT. For those subjects whose first "Dispdate" preceded the first PFT date, their first "Dispdate" was subtracted from the date of their first PFT, with the result indicative of the duration of potential dust exposure prior to the first PFT.
>
> iii) Thirdly, for some subjects, the date of their first x-ray did not correspond with the date of their first recorded PFT. Although for some of these subjects the first "Dispdate" and first x-ray date corresponded, in many cases they did not. For those subjects whose first x-ray date preceded the first PFT date, their first x-ray date was subtracted from the date of their first PFT, with the result indicative of the duration of potential dust exposure prior to the first PFT.

In order to consolidate the above three data sources, the source with the maximum value for duration of potential dust exposure prior to the first PFT was used. This value was designated as "**previous exposure**" in regression analyses.

A key assumption was that the occupational exposure history duration value obtained from the "Master" file corresponded to the number of years of dust exposure prior to the first PFT (as opposed to the first "Dispdate" or first x-ray date). For example, if the "Master" file occupational history for a subject was 30 years and the first "Dispdate" occurred 10 years before the first PFT date, then the duration of potential dust exposure prior to the first PFT for that subject would be 30 years, not 40 years.

There was no way to completely confirm that this assumption was accurate based on the available data. Visual inspection was performed for cases with duration values greater than "0" years for both "Master" file occupational history and a first date discrepancy (ie ii or iii, above). In virtually all cases, adding the two exposure duration values together either resulted in a sum that exceeded the individual's age at the time of their first PFT (or indicated a very young age at first exposure), or the first date discrepancy value added little to the "Master" file occupational history (for example, "Master" file exposure duration of 30 years and date discrepancy duration of 2 years). Therefore, the assumption that the "Master" file occupational exposure history duration value corresponded to the number of years of dust exposure prior to the first PFT appeared reasonable.

For subjects with at least two PFTs, the duration of exposure between the first and last PFT was calculated by subtracting a subject's first PFT date from their last PFT date. This duration was then added to the exposure that occurred prior to the first PFT (determined as per above), and the total represented the duration of dust exposure that occurred prior to the last Fibrosis Program PFT, and was designated "**exposure duration**" in regression analyses.

3.3.4 Age

Subject age at the time of PFT was determined by subtracting the subject's date of birth from the date of the PFT.

3.3.5 Sex

The subjects in the Fibrosis Program database were predominantly male, and both male and female subjects were included in analyses. For the regression analyses, males were coded as "1" and females were coded as "2". Therefore, a negative regression coefficient for the independent variable "sex" indicated that the dependent variable value was lower in females than in males.

3.3.6 Height

All height values were expressed in centimetres. The vast majority of subjects with any height information had only a single entry for height out of all of their PFTs, usually their first PFT. Subject height data was only included in analyses if one of two criteria were met: a) the subject had a single height value; b) the subject had more than one height value and the standard deviation of the height values was less than 2 cm. For criterion (a) the single value was used as the subject's height, whereas for criterion (b) the mean of the height values was used as the subject's height. Only 1-2% of subjects with any height information did not satisfy either criterion, depending on the analysis.

3.3.7 Symptoms and Past Medical History

In the original "Master" file, specific symptoms and past illnesses were coded as either a "1" or "2", which was presumed to indicate either the presence or absence of a specific symptom or past illness. Unfortunately, approximately 97% of subjects had missing symptom or past illness data, and so these variables were not considered further in the analyses.

3.4 Subjects Available for Analysis

There were 29515 subjects within the original "PFTdata" file. The processing steps involved in reducing the dataset to the subjects that were included in the analysis are summarized in Figure 2, and described in more detail below.

A small number of subject identification numbers in the "PFTdata" file had duplicate entries in the "Master" file, but these entries had different names, dates of birth, and other information for the same identification number. Deletion of these file numbers and subjects whose first PFT date occurred before 1964 (the year of the first Alberta survey of dust-exposed workers) reduced the number of subjects to 29237 from 29515.

Although all subjects in the "PFTdata" file had corresponding PFT dates, a sizeable proportion of subjects did not have any values entered into the PFT outcome fields observed FEV_1 and observed FVC. The exclusion of subjects without any observed PFT outcome values reduced the dataset to 7848 subjects. It was noted that in some PFT entries, the FEV_1 exceeded the FVC, a physiologic impossibility. After exclusion of these PFT entries and removal of four outliers with extreme FVC values, the data set was further reduced to 7763 subjects. Of these, 3895 subjects had height data. Data from these 3895 subjects were used in further analyses that examined the effects of dust exposure prior to the first PFT (see Section 3.5.2).

Of the 7763 subjects with at least one recorded PFT and height data, 3316 subjects had only a single PFT entry. This left 4447 subjects potentially available for longitudinal analyses that compared changes in PFT outcomes over time. For the majority (roughly 75%) of the 4447 subjects with two or more PFTs, the exposure code was the same for all of their PFT entries, indicating a single type of dust exposure for the duration of their enrolment in the Fibrosis Program. For these subjects, the PFT with the latest date was defined as their last PFT.

The last PFT was defined differently for those subjects who had at least one change in exposure code over the course of their PFT examinations. In order to allow for comparisons between individuals with only one type of dust exposure, the exposure code that corresponded to the initial PFT for each subject was defined as the main type of dust exposure. For subsequent PFTs, the first PFT with a corresponding exposure code that differed from the main type of dust exposure was identified. The previous PFT (the last PFT with the same exposure code as the main type of dust exposure) was then defined as the last PFT for that particular subject. For example, a subject whose first 4 PFTs were coded as "coal" and the subsequent 2 PFTs coded as "asbestos" would be classified as a "coal-exposed worker" and their fourth PFT would be become their last PFT for analysis purposes.

After this data processing step, there were 4206 subjects. This number was smaller than the 4447 total subjects with two or more PFTs and height data due to subject losses from the recoding of last PFTs for subjects with more than one type of dust exposure (if the exposure code for the second PFT was different than the main exposure type, then that subject would only have one PFT of the main exposure type and so would not be included in longitudinal analyses). Of these 4206 subjects, 2425 had height data. Data from these 2425 subjects were used in further analyses that examined the effects of dust exposure prior to the last PFT (see Section 3.5.3).

And finally, a subset of 1970 subjects with two or more PFTs had at least 10 years duration of exposure prior to their last PFT. Of these 1970 subjects, 1286 had height data. Data from these 1286 subjects were used in further analyses (see Section 3.5.4).

3.5 Analysis Steps

Unfortunately, the Fibrosis Program database was limited for a number of reasons. Chief among these was the small proportion of subjects in the "PFTdata" file who had observed FEV_1 and FVC values. Only 7763 subjects out of 29 237 (27%) had a least one recorded PFT. For longitudinal analyses, only 4206 subjects had at least two recorded PFTs, 14% of the subjects in the original "PFTdata" file. These small subject numbers call into question the representativeness of the analyzed subjects as compared to all subjects in the Fibrosis Program. Another limitation of the database was a lack of data on potentially important confounding variables such as smoking.

With these limitations in mind, the intent of the analysis was to: explore the representativeness of the analyzed subject samples; conduct analyses that examined the effects of dust exposure on first PFT outcomes, both within and between dust exposure groups; conduct analyses that examined the effects of dust exposure on last PFT outcomes, both within and between dust exposure groups. The analysis steps are outlined below.

3.5.1 All Subjects (n = 29237)

Analyses conducted on all subjects were descriptive in nature and intended to note key features of the entire "PFTdata" cohort.

3.5.2 Analyses of First PFT Outcomes for Subjects with at Least One PFT

A total of 7763 subjects had at least one PFT, but regression analyses were restricted to those 3895 subjects with height data.

Simple linear regression analyses were performed to determine the relationship between individual independent variables and the dependent variables of interest: FEV_1, FVC, and FEV_1/FVC%. Significant covariates included age, sex, height, date of PFT, previous exposure, and exposure code.

3.5.2.1 Analyses Within Exposure Groups

The influence of previous dust exposure on first PFT outcome variables was examined using simple linear regression for all subjects, and then with separate analyses restricted to coal-exposed subjects, silica/other dust-exposed subjects, and asbestos-exposed subjects.

Multiple linear regression analyses, using age at the first PFT, sex, height, date of the first PFT, and previous exposure as independent variables were then performed for each dependent variable: FEV_1, FVC, and FEV_1/FVC% of the first PFT. These regression analyses were performed for all subjects, and then repeated in separate analyses restricted to coal-exposed subjects, silica/other dust-exposed subjects, and asbestos-exposed subjects.

Multiple linear regression analyses, using date of the first PFT and previous exposure as independent variables, were then performed for each dependent variable: percent-predicted FEV_1 and percent-predicted FVC of the first PFT. These regression analyses were performed for all subjects, and then repeated in separate analyses restricted to coal-exposed subjects, silica/other dust-exposed subjects, and asbestos-exposed subjects.

3.5.2.2 Analyses Between Exposure Groups

In order to compare dust exposure groups, indicator variables were created for each dust exposure type. For example, to examine the effects of coal exposure, the coal indicator variable was set to "1" for all subjects whose exposure type was coal and the variable was set to "0" for all other subjects (whose exposure was either silica/other dust, asbestos, or MMMF). The regression coefficient for this indicator variable would then represent the difference in the PFT parameter between subjects whose main exposure was coal and all other subjects. A positive coefficient would indicate that the PFT parameter (eg FVC) was greater in the coal-exposed subjects as compared to the subjects with other types of dust exposure. The regression analysis would then be repeated by instead using a silica/other dust indicator variable (with a value of 1 for subjects whose main exposure type was silica/other dust and a value of 0 for all other subjects), and repeated again using an asbestos indicator variable in the same manner. Overall, there were 4 main dust exposure types: coal, silica/other dust, asbestos, and MMMF. Because of low subject numbers in the MMMF group, indicator variables were created for coal, silica/other dust, and asbestos only; subjects whose main dust exposure type was MMMF were still included with "all other subjects".

Multiple regression analyses, using the independent variables age at the first PFT, sex, height, date of the first PFT, and previous exposure, were performed for each dependent variable: FEV_1, FVC, and FEV_1/FVC% of the first PFT. These regressions were repeated three times, each time with a different exposure type indicator variable, as discussed above. Subjects without an exposure code were excluded. This process was repeated for percent-predicted FEV_1 and FVC, with age, sex, and height removed as independent variables from the regression model.

3.5.3 Analyses of Last PFT Outcomes for Subjects with Two or More PFTs

The null hypothesis of this study was that pulmonary function parameters at last contact with the Fibrosis Program would not differ significantly between workers exposed to coal, silica/other dust, or asbestos. The key dependent variables of interest to test this hypothesis were the results of the last PFT performed on each subject as part of the Fibrosis Program.

A total of 4206 subjects had two or more PFTs, but regression analyses were restricted to those 2425 subjects with height data.

Simple linear regression analyses were performed to determine the relationship between individual independent variables and the dependent variables of interest: FEV_1, FVC, and FEV_1/FVC% of the last PFT. Significant covariates included the first PFT value, age at the last PFT, sex, height, date of the last PFT, exposure duration, and exposure type.

3.5.3.1 Analyses Within Exposure Groups

The influence of previous dust exposure on the last PFT outcome variables was examined using simple linear regression for all subjects, and then with separate analyses restricted to coal-exposed subjects, silica/other dust-exposed subjects, and asbestos-exposed subjects.

Multiple linear regression analyses, using the first PFT value, age at the last PFT, sex, height, date of the last PFT, and exposure duration as independent variables were then performed for each dependent variable: FEV_1, FVC, and FEV_1/FVC% of the last PFT. These regression analyses were performed for all subjects, and then repeated in separate analyses restricted to coal-exposed subjects, silica/other dust-exposed subjects, and asbestos-exposed subjects.

Multiple linear regression analyses, using the first PFT value, date of the last PFT, and exposure duration as independent variables, were then performed for each dependent variable: percent-predicted FEV_1 and percent-predicted FVC of the last PFT. These regression analyses were performed for all subjects, and then repeated in separate analyses restricted to coal-exposed subjects, silica/other dust-exposed subjects, and asbestos-exposed subjects.

3.5.3.2 Analyses Between Exposure Groups

Dust exposure groups were compared in regression analyses that used indicator variables created for each dust exposure type (described in more detail in Section 3.5.2.2).

Multiple regression analyses, using the independent variables first PFT value, age at the last PFT, sex, height, date of the last PFT, and exposure duration, were performed for each dependent variable: FEV_1, FVC, and FEV_1/FVC% of the last PFT. These regressions were repeated three times, each time with a different exposure type indicator variable, as discussed in Section 3.5.2.2. Subjects without an exposure code were excluded. This process was repeated for percent-predicted FEV_1 and FVC of the last PFT, with age at the last PFT, sex, and height removed as independent variables from the regression model.

3.5.4 Analyses of Last PFT Outcomes for Subjects with Two or More PFTs and Ten or More Years Exposure Duration

The vast majority of pulmonary function studies of dust-exposed workers have observed that declines in pulmonary function attributable to dust exposure are typically not seen until after approximately ten years of dust exposure. Therefore, in order to optimize the chances of observing effects on pulmonary function secondary to dust exposure, the analyses described in Section 3.5.3 were repeated on the subset of subjects (n = 1703) who had ten or more years exposure duration. A total of 1970 subjects made up this subset, but regression analyses were restricted to those 1286 subjects with height data.

3.5.5 X-Ray Analyses

Subjects who had at least one x-ray with an assigned ILO code were identified. Radiographic pneumoconiosis was defined as an ILO code of 1/0 or greater. Subjects with at least one ILO coded x-ray were divided into three groups: 1) subjects for whom all of their x-rays were coded < 1/0 ("normal"); 2) subjects whose initial x-ray at entry into the Fibrosis program was < 1/0, but who had at least one subsequent x-ray coded ≥ 1/0 ("incident pneumoconiosis"); and 3) subjects whose initial x-ray at entry into the Fibrosis program was ≥ 1/0 ("pre-existing pneumoconiosis").

To assess the duration of exposure prior to the identification of pneumoconiosis, time since first entry into Fibrosis Program as well as occupational exposure history from the "Master" file (dust exposure prior to entry into the Fibrosis Program) were considered. The date of the first x-ray, first PFT, and first "Dispdate" were each separately subtracted from the date of the first x-ray coded as 1/0 or greater for each subject. Of those 3 separate subtractions, the one that gave the greatest value represented the duration of exposure from the time of entry into the Fibrosis Program to the identification of pneumoconiosis. This value was then added to the occupational exposure history value from the "Master" file for each subject to give a total exposure history for each subject. This total represented the time, in years, from first documented exposure to the identification of pneumoconiosis. For the purposes of addition, missing data for occupational exposure history from the "Master" file were given a value of zero.

For analyses of the influence of pneumoconiosis on pulmonary function, all exposure groups were included together due to limited subject numbers. Regression analyses in this section included an indicator variable for the presence of pneumoconiosis. Subjects with either incident or pre-existing pneumoconiosis were coded as "1", while subjects with normal x-rays were coded as "0". Subjects without any x-ray data were excluded from regression analyses.

3.5.6 Smoking Analyses

Smoking information was contained within the "Dispdata" file. Although this file contained multiple data entries for each worker that presumably corresponded to each examination that the worker attended, smoking data, if present, typically only appeared in one of the entries. Therefore, when available, smoking data represented smoking status for a subject at only one point in time. Although a data column existed for number of years smoked, less than 15% of subjects with any smoking data had a value entered into this column. Because of these low subject numbers, duration of smoking was not considered in analyses.

Smoking status was grouped broadly into four categories: present smokers, ex-smokers, never smokers, and "ambiguous" smokers. "Ambiguous" smokers were subjects who were classified in the data file as both present and ex-smokers. Group means were compared using a one-way ANOVA.

In order to maximize subject numbers for analyses, a simpler categorization was also used: ever smokers (present-smokers + ex-smokers + ambiguous smokers) and never smokers. The means for these categories were compared using an Independent Samples T-Test.

In order to assess the influence of smoking on the relationship between dust exposure and lung function, smoking was included in multiple linear regression analyses for all subjects, and then with separate analyses restricted to coal-exposed subjects, silica/other dust-exposed subjects, and asbestos-exposed subjects. For the smoking variable, ever smokers were coded as "1", and never smokers were coded as "2"; a positive regression coefficient for the independent variable "smoking" indicated that the dependent variable value was greater in non-smokers. Because of the low subject numbers involved, the coefficients obtained from including smoking in the regression models would not be directly comparable to the previous within exposure group comparisons (Section 3.5.2.1 and Section 3.5.3.1). Therefore, in order to assess the effect of smoking on the relationship between dust exposure and lung function, the regressions were repeated both with and without smoking in the regression model on the same groups of subjects.

3.6 Statistical methods

All statistical analyses were performed using SPSS.

Descriptive comparisons were made using Independent Samples T-Test for continuous variables (such as age, date of PFT, etc.) and Chi-square test for categorical variables (such as sex, exposure type, etc.). The null hypothesis for these statistical tests was that the compared parameters would not differ between groups. Significant differences were those with a p value less than 0.05, using a two-tailed test of significance.

As described above, the main regression analyses were performed to assess the effects of dust exposure on pulmonary function both within and between dust exposure groups.

For within exposure group comparisons, four sets of regression analyses were carried out, different only by the included subjects: all subjects, restricted to coal-exposed subjects only, restricted to silica/other dust-exposed subjects only, and restricted to asbestos-exposed subjects only. Within each of these four sets, separate regression analyses were performed for each pulmonary function dependent variable. The key independent variable of interest for the within exposure group regression analyses was duration of exposure ("previous exposure" for analyses of the first PFT, and "exposure duration" for analyses of the last PFT).

For between exposure group comparisons, three sets of regression analyses were carried out, and each set included all exposure groups. The only difference between these three sets of analyses was the specific exposure code indicator variable used: the first set of regression analyses compared coal-exposed subjects to all other subjects, the second set compared silica/other dust-exposed subjects to all other subjects, and the third set compared asbestos-exposed subjects to all other subjects. Within each of these three sets, separate regression analyses were performed for each pulmonary function dependent variable. The key independent variable of interest for the between exposure group regression analyses was the exposure code indicator variable ("coal", silica/other dust", or "asbestos").

One-way ANOVA was used for descriptive comparisons and also to compare percent-predicted FEV_1 and FVC between exposure groups. Scheffe's test was used for post hoc comparisons. The Bonferroni correction was used such that the level of significance for post hoc comparisons between three groups of subjects was 0.017 and for four groups of subjects was 0.008.

3.7 Ethical Approval

The protocol for this study was approved by the Health Research Ethics Board of the University of Alberta.

4.0 Results

4.1 All subjects (n = 29237)

4.1.1 Enrolment

Legislation concerning the Fibrosis Program came into effect in Alberta on July 1, 1966, but the first survey of lung health of workers in dusty trades took place in 1964. Active surveillance through central government assimilation and storage of Fibrosis Program records ended in 1997.

The entry of subjects into the Fibrosis Program, including the initial 1964 survey, is depicted graphically in Figure 3. As illustrated, the bulk of subject enrolment into the Fibrosis Program occurred in the mid-1970's and early 1980's, with peak enrolment in 1982. From 1964 to 1974, 5980 subjects (20% of the total) were enrolled in the Fibrosis Program, increasing to 17582 subjects (60% of the total) from 1975 to 1984. Enrolment dropped off sharply after 1986.

Enrolment of subjects with PFTs appeared to be somewhat biphasic, with relatively higher periods of enrolment for these subjects in the late-1970s and after 1990, although this trend was less pronounced for subjects with two or more PFTs.

Figures 4 to 6 graphically depict subject enrolment for the main dust exposure groups: coal, silica/other dust, and asbestos. Among subjects with PFTs, trends for each specific dust exposure group are similar to the PFT cohort as a whole, with somewhat biphasic periods of higher enrolment in the late-1970s and after 1990. However, for asbestos-exposed subjects, the post-1990 rise in enrolment is more attenuated as compared to the other dust exposure groups.

4.1.2 Missing Data

As noted in the Methods section, some of the variables contained within the original Fibrosis Program data files did not contain complete information for all subjects. Most notably for the purposes of this analysis, only 7763 subjects (27%) had any PFT outcome data. Subjects with available information for other variables are provided in Table 4, divided into PFT groups: subjects without PFT information, subjects with at least one PFT, and subjects with two or more PFTs.

4.1.3 Industry Representation

The frequencies of the different industry categories assigned to subjects at enrolment into the Fibrosis Program are provided in Table 5. The most frequent industry category was "coal mining", ranging from 41% of subjects without PFT information to 49% of subjects with two or more PFTs. For all major subject groupings, the top four most common industry categories were "Coal Mining", "Insulation Company", "Quarries, Gravel", and "Government of Alberta or Municipalities, Hospitals", which together made up about 80% of all industry categories.

Exposure codes assigned to subjects, as a function of industry category, are provided in Figure 7. The predominant industry code for coal-exposed subjects was "Coal Mining" (96% of coal-exposed subjects). The predominant industry code for silica/other dust-exposed subjects was "Government of Alberta or Municipalities, Hospitals" (50% of silica/other-dust-exposed subjects). The predominant industry code for asbestos-exposed subjects was "Insulation Company" (71% of asbestos-exposed subjects).

4.2 Analyses of First PFT Outcomes for Subjects with at Least One PFT

4.2.1 Descriptive comparisons

A total of 7763 subjects had at least one PFT, and 3895 of those subjects also had height data. Subjects with at least one PFT, with and without height data, are compared with remaining subjects in Tables 6a and 6b, respectively. There were no significant differences in the proportion of male and female subjects. Subjects with at least one PFT and height data enrolled in the Fibrosis Program at a considerably later date as compared to subjects who either had no PFT data or no height data. The proportion of subjects with an exposure code was about 96%, although this was slightly lower for subjects with at least one PFT and height data. The order of the exposure groups in terms of subject proportions was similar between compared groups: the most common exposure was to coal, followed by silica/other dust, asbestos, and then MMMF. However, the PFT group, with and without height data, had proportionately more subjects with coal and MMMF exposure and fewer subjects with silica/other dust and asbestos exposure than remaining subjects.

Descriptive comparisons were made between exposure groups for subjects with at least one PFT, and the results are presented in Table 7. The greatest proportion of female subjects is observed in the coal-exposed group. Subjects in the silica/other dust group on average were older at enrolment and enrolled later.

Over one fifth of subjects had some degree of previous dust exposure prior to the first Fibrosis Program PFT, and this proportion was greatest for subjects with asbestos exposure, at 29%. For those subjects with previous exposure, the average duration of that exposure was significantly greater for the silica/other dust-exposed subjects.

There were no differences across exposure groups for mean FEV_1, but FVC was significantly greater for the silica/other dust exposure group. Modest but significant differences existed between exposure groups for $FEV_1/FVC\%$.

Silica/other dust-exposed subjects were more likely to have height data, but among subjects with height data, there were no significant mean height differences across exposure groups. Among the subset of subjects with height data, there were no significant differences in the proportion of subjects with previous dust exposure across exposure groups. For those subjects with previous exposure, the average duration of that exposure was significantly shorter for coal-exposed subjects. With respect to PFT outcomes in the subset of subjects with height data, asbestos-exposed subjects had a significantly greater FEV_1 than silica/other dust-exposed subjects, and a significantly greater $FEV_1/FVC\%$ as compared to both coal and silica/other dust-exposed subjects.

4.2.2 Simple Linear Regression

Regression analyses were restricted to the 3895 subjects with height data.

Simple linear regression analyses were performed to examine the relationship between independent variables and the first PFT outcome variables: FEV_1, FVC, and $FEV_1/FVC\%$. The results are summarized in Table 8. As expected, there was an inverse relationship between age and FEV_1, FVC, and $FEV_1/FVC\%$, with each year of age accounting for a drop in FEV_1 and FVC of 25 mL and 21 mL, respectively. On average, females had lower FEV_1 and FVC values than males. The year of the PFT examination had a positive relationship with FEV_1 and FVC, indicating that enrolment PFT values increased over the life of the Fibrosis Program. Among subjects with height data, the positive relationship between height and both FEV_1 and FVC was as expected.

There was no significant difference in PFT parameters between coal-exposed subjects and subjects with other dust exposure types. Silica/other dust-exposed subjects had significantly lower FEV_1, FVC, and $FEV_1/FVC\%$ than other subjects by 96 mL, 65 mL, and 0.9%, respectively. The FEV_1 of asbestos-exposed subjects was 130 mL greater and their $FEV_1/FVC\%$ was 2.3% greater, on average, than subjects with other dust exposure types.

4.2.3 Within Exposure Group Analyses

Table 9 summarizes the results of simple linear regression analyses for the independent variable of previous dust exposure for each type of dust exposure. When all subjects were considered, each year of previous dust exposure was associated with a 13 mL decline in both FEV_1 and FVC, and a 0.05% decline in $FEV_1/FVC\%$. When the analysis was restricted to specific dust types, the largest effect of previous dust exposure on pulmonary function appeared to be limited to silica/other dust-exposed subjects, who had a 22 mL, 21 mL, and 0.09% decline in FEV_1, FVC, and $FEV_1/FVC\%$, respectively, with each year of dust exposure prior to the first PFT. The only other significant dust-specific relationship of note was a 9 mL decline in FEV_1 per year of previous dust exposure for coal-exposed subjects.

The independent variable of previous dust exposure was then combined with age, sex, height, and first PFT date in multiple linear regression analyses, summarized in Table 10. The inclusion of other covariates in the regression model attenuated the effects of previous dust exposure on pulmonary function parameters. However, previous dust exposure still had a significant relationship with PFT performance for silica/other dust-exposed subjects, with a 9 mL decline in FEV_1 and 0.09% decline in FEV_1/FVC% for each year of dust exposure prior to the first PFT. There were no significant relationships between previous exposure and pulmonary function for coal-exposed subjects, asbestos-exposed subjects, or all subjects combined.

The results of multiple linear regression analyses for the dependent variables percent-predicted FEV_1 and FVC are provided in Table 11. The only significant relationship between previous dust exposure and pulmonary function is again found only in the silica/other dust-exposed group, with each year of dust exposure prior to the first PFT associated with a 0.19% decline in percent-predicted FEV_1.

4.2.4 Between Exposure Group Analyses

Multiple linear regression analyses were performed on all subjects with an exposure code three times, each with a different exposure type indicator variable. Results are provided in Table 12. As compared to other dust-exposed subjects, silica/other dust-exposed subjects had a 69 mL lower FEV_1 and a 61 mL lower FVC. In contrast, asbestos-exposed subjects had considerably greater PFT values than subjects with other types of dust exposure. Asbestos-exposed subjects had a 152 mL greater FEV_1, a 2.1% greater FEV_1/FVC%, both significant, and a 65 mL greater FVC that approached statistical significance ($p = 0.055$).

A similar pattern is observed for percent-predicted FEV_1 and FVC, as displayed in Table 13. Silica/other dust-exposed subjects had a 1.5% and 1.1% lower percent-predicted FEV_1 and FVC, respectively, whereas asbestos-exposed subjects had greater values, although only the 3.4% greater percent-predicted FEV_1 reached significance.

Mean percent-predicted FEV_1 and FVC were also compared using a one-way ANOVA (Table 14), which did not account for previous exposure or PFT date. Although there were no significant differences for percent-predicted FVC, percent-predicted FEV_1 for the asbestos group was significantly greater than the corresponding value for the coal group, by 2.5%. Although this difference is statistically significant, it is likely not clinically significant, given that the percent-predicted values closely matched population-derived expected values.

4.2.5 Between Exposure Group Analyses, No Previous Dust Exposure

The above regressions were also repeated for the subset of 2787 subjects (1387 coal-exposed, 985 silica/other dust-exposed, 393 asbestos-exposed, and 22 MMMF-exposed) with zero years of previous exposure. Previous exposure was removed as a variable in the regression model, but all other regression variables remained the same. In this analysis, none of the PFT parameters of silica/other dust-exposed subjects were significantly different as compared to subjects with other dust exposure types (Exposure type regression coefficients were - 25 mL, - 44 mL and 0.188% for FEV_1, FVC, and FEV_1/FVC%, respectively). Coal exposed subjects had a 55 mL lower FEV_1 and 1.2% lower FEV_1/FVC%, while asbestos-exposed subjects had a 151 mL greater FEV_1 and 2.0% greater FEV_1/FVC%).

These results were mirrored in the regression analyses for percent-predicted FEV_1 and FVC. These PFT parameters were not significantly different for silica/other dust-exposed subjects as compared to subjects with other dust exposure types (Exposure type regression coefficients were – 0.226% and – 0.678% for percent-predicted FEV_1 and FVC, respectively). Coal exposed subjects had a 1.441% lower percent-predicted FEV_1, while asbestos-exposed subjects had a 3.275% greater percent-predicted FEV_1.

4.2.6 Analysis Summary of First PFT Outcomes for Subjects with at Least One PFT

At the time of the first Fibrosis Program PFT, roughly a quarter of subjects had at least some degree of previous dust exposure. In both simple linear regression and multiple linear regression analyses that examined dust exposure groups separately, previous dust exposure had a consistently deleterious effect on lung function for silica/other dust-exposed subjects. Previous exposure had little to no significant effect on pulmonary function for coal or asbestos-exposed subjects.

The poorer performance of silica/other dust-exposed subjects was again observed when all subjects were examined together; silica/other dust-exposed subjects had a modest but significantly lower FEV_1, FVC, percent-predicted FEV_1, and percent-predicted FVC as compared to subjects with other dust exposure types. Asbestos-exposed subjects had consistently better PFT performance than subjects with other dust exposure types.

In the subset of subjects with no documented previous dust exposure prior to their first PFT, the relatively greater PFT performance of asbestos-exposed subjects was also evident. However, the previously observed relatively poorer performance of silica/other dust-exposed subjects was no longer apparent, and their pulmonary function was not significantly different than subjects with other dust exposure types. As well, when compared to subjects with other dust exposure types, coal-exposed subjects had somewhat poorer pulmonary function.

4.3 Analyses of Last PFT Outcomes for Subjects with Two or More PFTs

4.3.1 Descriptive Comparisons

Of the 7763 subjects with at least one PFT, 4206 subjects had two or more PFTs. Descriptive comparisons between subjects with two or more PFTs and the remaining subjects with PFT data are presented in Table 15. Subjects with two or more PFTs had proportionately more male subjects. As well, despite enrolling in the Fibrosis Program earlier, subjects with two or more PFTs were not significantly different in age than subjects with less than two PFTs. Subjects with two or more PFTs were more likely to have exposure code information. The order of the exposure groups in terms of subject proportions was similar between the two groups: the most common exposure was to coal, followed by silica/other dust, asbestos, and then MMMF. However, the subjects with two or more PFTs had proportionately more subjects with coal and fewer subjects with silica/other dust exposure than subjects with less than two PFTs.

A greater proportion of subjects with two or more PFTs had previous dust exposure prior to the first PFT as compared to subjects with less than 2 PFTs. For those subjects with previous dust exposure, there was no significant mean difference in the duration of that exposure between subjects with two or more PFTs and subjects with less than two PFTs.

There were no significant differences in mean PFT values between the subjects with two or more PFTs and the subjects with less than two PFTs, when all subjects were considered.

Subjects with two or more PFTs were more likely to have height data than subjects with less than two PFTs, although there was no significant difference in mean height between these groups. For the subset of subjects with height data, mean FVC was lower and mean FEV_1/FVC% was greater for subjects with two or more PFTs. As well, mean percent-predicted FEV_1 and FVC were significantly lower for the subjects with two or more PFTs.

Table 16 summarizes comparisons between different exposure groups for subjects with two or more PFTs. The proportion of male and female subjects was similar across exposure groups. Subjects in the silica/other dust group were significantly older than subjects in both the coal and asbestos groups at both the first and last PFTs. Subjects in the coal exposure group were more likely to have height data and on average, were taller than subjects in the silica/other dust and asbestos exposure groups.

When all subjects with two or more PFTs were considered, silica/other dust-exposed subjects had a mean time between first and last PFTs of 8.4 years, which was slightly shorter than the equivalent period for coal and asbestos-exposed subjects. However, when exposure prior to the first PFT was included with this time period (ie "exposure duration"), there were no significant differences between dust exposure groups. Mean exposure duration ranged from 10.5 years for silica/other dust-exposed subjects to 11.0 years for asbestos-exposed subjects. The proportion of subjects with any degree of previous dust exposure was not significantly different between dust exposure groups, ranging between 27% and 31%. For those subjects with previous dust exposure prior to the first PFT, silica/other dust-exposed subjects had a mean duration of previous exposure of 7.6 years, which was significantly greater then the other dust exposure groups.

PFT parameters across exposure groups for subjects with two or more PFTs are summarized in Table 17. When all subjects were considered, there were no significant differences for FEV_1 at the first PFT between exposure groups. The 137 mL difference in mean FVC values between coal-exposed and asbestos-exposed subjects reached statistical significance. Subjects in the asbestos exposure group also had a first FEV_1/FVC% that was significantly greater than the other exposure groups. At the last PFT, coal-exposed subjects had an 87 mL greater FEV_1 and a 135 mL greater FVC as compared to silica/other dust-exposed subjects and asbestos-exposed subjects, respectively.

PFT parameters for the subset of subjects with height data are also compared in Table 17. At the first PFT, silica/other dust-exposed subjects had a significantly lower FEV_1 as compared to coal and asbestos-exposed subjects, and a significantly lower FVC as compared to coal-exposed subjects. The first FEV_1/FVC% of asbestos-exposed subjects was almost 3% greater, on average, than the corresponding value of coal and silica/other dust-exposed subjects. At the last PFT, there were no significant differences between different dust exposure groups for any PFT parameter.

4.3.2 Simple Linear Regression

Regression analyses were restricted to the 2425 subjects with height data.

Simple linear regression analyses were performed to examine the relationship between independent variables and the last PFT outcome variables: FEV_1, FVC, and FEV_1/FVC%. The results are summarized in Table 18. Regression coefficients for sex and height were virtually identical to the simple linear regressions performed for the first PFT (Table 8). The date of last PFT coefficients were positive, indicating an improvement in pulmonary function at the last PFT over the life of the Fibrosis Program, although these values were slightly attenuated from the first PFT analyses (Table 8). As a lone variable, each year of age was associated with a 36 mL decline in FEV_1 and 39 mL decline in FVC. As expected, PFT parameters at the first PFT were strongly associated with PFT parameters at the last PFT.

The only significant coefficients for exposure type were FEV_1 for silica/other dust-exposed subjects (- 77 mL) and FEV_1/FVC% for asbestos-exposed subjects (+ 1.0 %). Otherwise, there were no significant differences when comparing any specific dust type to all other types of dust exposure.

4.3.3 Within Exposure Group Analyses

Table 19 summarizes the results of simple linear regression analyses for the independent variable of exposure duration for each type of dust exposure. When all subjects were considered, each year of previous dust exposure was associated with an 18 mL decline in both FEV_1 and FVC, and a 0.08% decline in FEV_1/FVC%. Exposure duration had a significantly negative effect on almost all last PFT parameters when the analysis was restricted to specific dust types. The largest coefficients were observed in coal-exposed subjects, with each year of dust exposure associated with an average decline in FEV_1, FVC, and FEV_1/FVC% of 24 mL, 23 mL, and 0.1%, respectively. In terms of coefficient magnitude, asbestos-exposed subjects came next (- 19 mL, - 19 mL, -0.07% for FEV_1, FVC, and FEV_1/FVC%, respectively, per year of dust exposure). Interestingly, for the silica/other dust group the effects of dust exposure had an approximately 10 mL per year lesser impact on the last PFT FEV_1 and FVC as compared to the effects of dust exposure on these outcome measures at the first PFT (Table 9)

The independent variable of exposure duration was then combined with the corresponding first PFT value, age, sex, height, and last PFT date in multiple linear regression analyses, summarized in Table 20. With the inclusion of other covariates in the regression model, dust exposure no longer had a significant relationship with any last PFT outcome variable for silica/other dust-exposed subjects. For coal-exposed subjects, each year of previous dust exposure was associated with a 9 mL increase in FVC and a 0.1% decrease in FEV_1/FVC%. For asbestos-exposed subjects, after controlling for age, sex, height, PFT date, and first PFT value, each year of dust exposure prior to the last PFT was associated with a 10 mL decline in FEV_1 and a 0.13% decline in FEV_1/FVC%.

The results of multiple linear regression analyses for the dependent variables percent-predicted FEV_1 and FVC of the last PFT are provided in Table 21. When all subjects were considered together, each year of dust exposure was associated with a 0.11% increase in percent-predicted FEV_1 and a 0.09% increase in percent-predicted FVC. Positive effects of dust exposure on percent-predicted FEV_1 and FVC were observed for both coal and silica-other dust-exposed subjects, with the relationship for coal-exposed subjects being of greatest magnitude and highly significant. For asbestos-exposed subjects, dust exposure had a detrimental effect on percent-predicted PFT values, although this only reached significance for percent-predicted FEV_1, at –0.16% per year of dust exposure.

4.3.4 Within Exposure Group Analyses, No Previous Dust Exposure

The above multiple linear regressions were also repeated for the subset of 1783 subjects (1016 coal-exposed, 520 silica/other dust-exposed, and 247 asbestos-exposed) with zero years of previous exposure. Exposure duration was removed as an independent variable and replaced with the time between the first and last PFT in the multiple linear regression. All other regression variables remained the same. In this analysis, none of the PFT parameters of silica/other dust-exposed subjects were significantly associated with time between the first and last PFT (regression coefficients were – 0.006 mL/year, -0.001 mL/year, and – 0.050 %/year for FEV_1, FVC, and FEV_1/FVC%, respectively). Each year of exposure between the first and last PFT was associated with an 18 mL gain in FEV_1 and a 0.187% decline in FEV_1/FVC% for coal-exposed subjects, and a 14 mL decline in FEV_1 and 0.231% decline in FEV_1/FVC% for asbestos-exposed subjects.

In the regression analyses for percent-predicted FEV_1 and FVC, relationships with exposure were either positive or not significant. Each year of exposure between the first and last PFT was significantly associated with a 0.471% and 0.465% increase in percent-predicted FEV_1 and FVC, respectively, for coal-exposed subjects. The only other significant relationship with time between the first and last PFT was a 0.552% rise in percent-predicted FEV_1 per year for silica/other dust-exposed subjects.

4.3.5 Between Exposure Group Analyses

Multiple linear regression analyses were performed on all subjects with an exposure code three times, each with a different exposure type indicator variable. Results are provided in Table 22. Coal-exposed subjects had a 57 mL lower FEV_1 and an 82 mL lower FVC at the last PFT as compared to subjects with other types of dust exposure. The only other significant effect of exposure type was a 75 mL greater FVC for silica/other dust-exposed subjects as compared to subjects with other types of dust exposure. For all dust types, exposure duration had a very modest non-significant negative effect on FEV_1 and a modest positive effect on FVC, reaching significance for coal and silica/other dust-exposed subjects (and approaching significance for asbestos-exposed subjects, $p = 0.066$). This resulted in a significant negative effect of exposure duration on FEV_1/FVC% of - 0.08% per year of dust exposure for all dust types.

The only significant relationships between dust type and percent-predicted FEV_1 and FVC at the last PFT were observed for coal-exposed subjects, who had values that were just over 1% lower than subjects with other dust exposure types (Table 23). Exposure duration had a significant positive relationship with percent-predicted PFT values for all exposure groups.

Mean percent-predicted FEV_1 and FVC for the last PFT were also compared using a one-way ANOVA (Table 24), which did not account for exposure duration or PFT date. There were no significant differences between exposure groups for mean percent-predicted FEV_1 or FVC at the last PFT. Interestingly, when compared to the mean values obtained from the first PFT for all subjects with PFT data (Table 14), the percent-predicted FEV_1 and FVC at the last PFT for subjects with two or more PFTs were around 1-2% higher.

4.3.6 Between Exposure Group Analyses, No Previous Dust Exposure

The above multiple linear regressions were also repeated for the subset of 1798 subjects (1016 coal-exposed, 520 silica/other dust-exposed, 247 asbestos-exposed, and 15 MMMF-exposed) with zero years of previous exposure. Exposure duration was removed as an independent variable and replaced with the time between the first and last PFT in the multiple linear regression. All other regression variables remained the same. In this analysis, negative coefficients for exposure type were observed for FEV_1 and FVC for coal-exposed subjects, and $FEV_1/FVC\%$ for silica/other dust-exposed subjects. All other exposure type regression coefficients were positive, but small and not significant. The FEV_1 and FVC of coal-exposed subjects was 35 mL (not significant) and 67 mL ($p < 0.05$) lower, respectively, than subjects with other dust exposure types. $FEV_1/FVC\%$ was 0.654% ($p < 0.05$) lower in silica/other dust-exposed subjects than subjects with other dust exposure types.

None of the exposure type regression coefficients for percent-predicted FEV_1 or FVC reached statistical significance. Coal-exposed subjects had a 0.625% and 0.773% lower percent-predicted FEV_1 and FVC, respectively, as compared to subjects with other dust exposure types, whereas asbestos-exposed subjects had a 1.324% and 0.890% greater percent-predicted FEV_1 and FVC, respectively. The corresponding values for silica/other dust-exposed subjects were 0.190% lower and 0.396% greater.

4.3.7 Analysis Summary of Last PFT Outcomes for Subjects with Two or More PFTs

In multiple linear regression analyses that examined dust exposure groups separately, the only significant associations between exposure duration and last PFT outcomes were a 9 mL increase in FVC per year and a 0.10% decrease in $FEV_1/FVC\%$ per year for coal-exposed subjects, and a 10 mL decrease in FEV_1 and 0.13% decrease in $FEV_1/FVC\%$ per year for asbestos-exposed subjects. There was no significant effect of exposure duration on the PFT parameters of silica/other dust-exposed subjects. For the subset of subjects with no history of dust exposure prior to their first PFT, similar relationships were observed in multiple linear regression analyses that used time between the first and last PFT as a variable: coal-exposed subjects had an increase in FVC and decline in $FEV_1/FVC\%$; silica/other dust–exposed subjects had no significant PFT parameter associations; and asbestos-exposed subjects had declines in FEV_1 and $FEV_1/FVC\%$ with each year of exposure between the first and last PFT.

Exposure duration had a positive effect on percent-predicted FEV_1 and FVC for coal and silica/other dust-exposed subjects. However, the opposite was true for asbestos-exposed subjects, with each year of exposure associated with a significant 0.158% decline in percent-predicted FEV_1 and a non-significant 0.123% decline in percent-predicted FVC.

When all subjects were examined together, coal-exposed subjects had modestly poorer pulmonary function at the last PFT as compared to subjects with other dust exposure types. After controlling for other independent variables, subjects with coal exposure had a 57 mL lower FEV_1 and an 82 mL lower FVC at the last PFT as compared to subjects with other types of dust exposure. The percent-predicted FEV_1 and FVC of coal-exposed subjects at the last PFT was just over 1% lower than subjects with other types of dust exposure.

In the subset of subjects with no documented previous dust exposure prior to their first PFT, the relatively poorer PFT performance of coal-exposed subjects as compared to subjects with other types of dust exposure was again evident. However, this was only significant for the 67 mL lower FVC of coal-exposed subjects.

4.4 Analyses of last PFT Outcomes for Subjects with Two or More PFTs and Ten or More Years Exposure Duration

4.4.1 Descriptive Statistics

Among subjects with two or more PFTs, 47% had ten or more years exposure duration. Descriptive comparisons of this group of subjects as compared to those subjects with two or more PFTs who had less than ten years exposure duration are provided in Table 25. On average, subjects with the longer exposure period were about three years younger at the time of their first PFT and their first PFT occurred about 6 years earlier than subjects with less than ten years exposure duration. Subjects with ten or more years exposure duration were also more likely to have height data.

The average exposure duration for subjects with at ten or more exposure duration was 17.3 years, versus 4.7 years for subjects with less than ten years exposure duration. Unlike subjects with less than ten years exposure duration, subjects with ten or more years exposure duration had a much higher proportion of subjects with dust exposure prior to their first PFT (45% versus 15%). As well, among subjects with dust exposure prior to their first PFT, subjects with ten or more years exposure duration also had a much greater exposure period prior to their first PFT (7.4 versus 1.8 years).

Mean PFT parameters for the first and last PFT are compared between exposure duration groups and across dust types in Table 26. Even though coal-exposed subjects with ten or more years between first and last PFTs were younger, on average, at the time of the first PFT, their mean first FEV_1 and FVC were 180 mL and 218 mL lower, respectively, than the corresponding values for subjects with less than ten years duration of exposure. This discrepancy was even greater for the subset of subjects with height data, with coal-exposed subjects with ten or more years exposure duration having mean FEV_1 and FVC values that were 370 mL and 503 mL lower, respectively, than coal-exposed subjects with less than ten years exposure duration at the time of the first PFT. By the time of the last PFT, the magnitude of the differences between exposure duration groups was attenuated somewhat for all coal subjects and those with height data, but all PFT parameters were still significantly lower in those coal-exposed subjects with ten or more years exposure duration.

An opposite first PFT trend was observed for the asbestos-exposed subjects. At the time of the first PFT, subjects in the asbestos group with ten or more years exposure duration had an approximately 200 mL greater mean FEV_1 and FVC than subjects with less than ten years exposure duration. By the time of the last PFT, these differences were no longer apparent. For the subset of asbestos-exposed subjects with height data, the only significant difference between exposure duration groups was a 162 mL lower FEV_1 at the last PFT for subjects with ten or more years exposure duration.

Mean percent-predicted FEV_1 and FVC values for the subset of subjects with height data are compared in Table 27. Mean first PFT percent-predicted FEV_1 and FVC are consistently significantly lower for subjects with ten or more years exposure duration for all dust exposure groups. Differences were greatest for coal-exposed subjects, at close to 10% for both percent-predicted FEV_1 and FVC, and more modest for silica/other dust and asbestos-exposed subjects at about 5% for both percent-predicted FEV_1 and FVC. For the last PFT, percent-predicted values were close to 3% lower for coal-exposed subjects at the time of the last PFT. For silica/other dust and asbestos-exposed subjects, last PFT percent-predicted values were not significantly different between subjects with ten or more years and less than ten years exposure duration.

4.4.2 Within Exposure Group Analyses

Table 28 summarizes the results of simple linear regression analyses for the independent variable of exposure duration for each type of dust exposure. When all subjects with ten or more years exposure duration were considered, each year of previous dust exposure was associated with a 14 mL decline in FEV_1 and a 18 mL decline in FVC. Unlike the simple linear regression analysis of all subjects with two or more PFTs (Table 19), there was no significant relationship between exposure duration and pulmonary function at the last PFT for coal-exposed subjects with at least ten years of exposure. For silica/other dust and asbestos-exposed subjects, exposure duration had a significantly negative impact on FEV_1 and FVC at the last PFT, ranging from a 19 mL decline in FEV_1 per year of dust exposure for silica/other dust-exposed subjects to a 27 mL decline in FVC per year of dust exposure for both silica/other dust and asbestos-exposed subjects.

Multiple linear regression analyses demonstrated a positive effect of dust exposure on last PFT pulmonary function for coal-exposed subjects and a negative effect for asbestos-exposed subjects (Table 29). Interestingly, the effects of age on the last PFT parameters ranged considerably across dust exposure groups. For each year of age: coal-exposed subjects experienced a 25 mL and 27 mL decline in FEV_1 and FVC, respectively; silica/other dust-exposed subjects experienced a 36 mL and 39 mL decline in FEV_1 and FVC, respectively; asbestos-exposed subjects experienced a 38 mL and 48 mL decline in FEV_1 and FVC, respectively. There were no significant effects of exposure duration on pulmonary function for silica/other dust-exposed subjects. For coal-exposed subjects with at least ten years of dust exposure, each year of exposure was associated with a 15 mL and 17 mL increase in FEV_1 and FVC, respectively. For asbestos-exposed subjects, each year of dust exposure was associated with a significant 12 mL decline in FEV_1, a 9 mL decline in FVC ($p = 0.060$), and a 0.1% decline in FEV_1/FVC% ($p = 0.051$).

Similar relationships were observed using percent-predicted FEV_1 and FVC of the last PFT as dependent variables (Table 30). Dust exposure had a significantly positive effect on percent-predicted pulmonary function values for coal-exposed subjects, a significantly negative effect for asbestos-exposed subjects, and no significant effect for silica/other dust-exposed subjects.

4.4.3 Within Exposure Group Analyses, No Previous Dust Exposure

The above multiple linear regressions were also repeated for the subset of 774 subjects (446 coal-exposed, 174 silica/other dust-exposed, and 124 asbestos-exposed) with zero years of previous exposure. Exposure duration was removed as an independent variable and replaced with the time between the first and last PFT in the multiple linear regression. All other regression variables remained the same. The results mirrored what was obtained when all subjects with ten or more years exposure duration were examined. Each year of dust exposure between the first and last PFT was associated with a significant 36 mL increase in FEV_1 and 58 mL increase in FVC for coal-exposed subjects, whereas asbestos-exposed subjects experienced a 50 mL and 41 mL decline in FEV_1 and FVC, respectively. The corresponding values for the silica/other dust-exposed subjects was a 6 mL increase in FEV_1 and 5 mL decrease in FVC, both non-significant.

Each year of dust exposure between the first and last PFT was associated with a significant 1.294% and 1.203% increase in percent-predicted FEV_1 and FVC, respectively, for coal-exposed subjects, whereas asbestos-exposed subjects had corresponding declines of 1.488% and 1.230%, respectively. The silica/other dust-exposed subjects had a non-significant increase in a percent-predicted FEV_1 of 0.317% and a non-significant decrease in percent-predicted FVC of 0.169%.

4.4.4 Between Exposure Group Analyses

Multiple linear regression analyses were performed on all subjects with an exposure code three times, each with a different exposure type indicator variable. Results are provided in Table 31. Although dust exposure appeared to have a positive effect on pulmonary function for coal-exposed subjects when they were examined on their own, coal-exposed subjects had poorer pulmonary function at the last PFT when compared to subjects with other types of dust exposure; in the between exposure group analysis, subjects exposed to coal dust for at least ten years had a FEV_1 and FVC that were 69 mL and 84 mL lower, respectively. As compared to subjects with other types of dust exposure, silica/other dust-exposed subjects had a significantly 81 mL greater FVC and a 63 mL greater FEV_1, which approached significance ($p = 0.061$). None of the PFT parameters of asbestos-exposed subjects were significantly different than those of subjects in other dust exposure groups.

The only significant relationships between dust type and percent-predicted FEV_1 and FVC at the last PFT were observed for coal-exposed subjects, who had values that were both 1.8% lower than subjects with other dust exposure types (Table 32).

Mean percent-predicted FEV_1 and FVC for the first and last PFT of subjects with ten or more years exposure duration were also compared using a one-way ANOVA (Table 33). The only significant mean difference between dust exposure groups was the 4% separation in percent-predicted FEV_1 of the first PFT for coal and asbestos-exposed subjects. All percent-predicted mean values were greater at the last PFT than at the first PFT. Increases in percent-predicted FEV_1 from the first PFT to the last PFT were greatest for silica/other dust-exposed subjects (7.0%) and lowest for asbestos-exposed subjects (4.6%). Increases in percent-predicted FVC from the first PFT to the last PFT were greatest for silica/other dust-exposed subjects (6.7%) and lowest for asbestos-exposed subjects (5.7%).

4.4.5 Between Exposure Group Analyses, No Previous Dust Exposure

The above multiple linear regressions were also repeated for the subset of 774 subjects (446 coal-exposed, 174 silica/other dust-exposed, and 124 asbestos-exposed) with zero years of previous exposure. Exposure duration was removed as an independent variable and replaced with the time between the first and last PFT in the multiple linear regression. All other regression variables remained the same. In this analysis, there were no significant differences in any PFT parameter when comparing any dust type against subjects with other dust exposure types. As compared to other subjects, coal-exposed subjects had a 45 mL, 33 mL, and 0.372% lower FEV_1, FVC, and $FEV_1/FVC\%$, respectively. As compared to other subjects, silica/other dust-exposed subjects had a 10 mL lower FEV_1, a 9 mL greater FVC and a 0.033% lower FEV_1/FVC%. Asbestos-exposed subjects had a 98 mL greater FEV_1 ($p = 0.053$), a 49 mL greater FVC, and a 0.748% greater FEV_1/FVC% as compared to subjects with other dust exposure types.

None of the exposure type regression coefficients for percent-predicted FEV_1 or FVC reached statistical significance. Coal-exposed subjects had a 0.864% and 0.434% lower percent-predicted FEV_1 and FVC, respectively, as compared to subjects with other dust exposure types; silica/other dust-exposed subjects had a 0.550% and 0.170% lower percent-predicted FEV_1 and FVC, respectively, as compared to subjects with other dust exposure types; asbestos-exposed subjects had a 2.380% ($p = 0.083$) and 1.041% greater percent-predicted FEV_1 and FVC, respectively, as compared to subjects with other dust exposure types

4.4.6 Analysis Summary of Last PFT Outcomes for Subjects with Two or More PFTs and Ten or More Years Exposure Duration

The most striking differences in first PFT parameters when comparing subjects with ten or more years exposure duration to subjects with less than ten years exposure duration were observed for coal-exposed subjects: subjects with ten or more years exposure duration had mean FEV_1 and FVC values that were several hundred millilitres lower. For all exposure groups, percent-predicted FEV_1 and FVC at the first PFT were 5-10% lower for subjects with ten or more years exposure duration than subjects with less than ten years exposure duration.

For within exposure group analyses, coal-exposed subjects demonstrated a consistently positive association between exposure duration and pulmonary function, whereas asbestos-exposed subjects demonstrated a consistently negative association. This was observed for PFT parameters expressed as a volume measurement and as a percentage of predicted. When only subjects with zero years of previous dust exposure prior to their first PFT were considered, exposure between the first and last PFT still had a positive effect on pulmonary function for coal-exposed subjects and a negative effect on pulmonary function for asbestos-exposed subjects.

Despite this apparent improvement in pulmonary function with exposure for coal-exposed subjects, when all subjects were examined together, coal-exposed subjects had modestly poorer pulmonary function at the last PFT as compared to subjects with other dust exposure types. After controlling for other independent variables, subjects with coal exposure had a 69 mL lower FEV_1 and an 84 mL lower FVC at the last PFT as compared to subjects with other types of dust exposure. They also had a 1.8% lower percent-predicted FEV_1 and FVC at the last PFT as compared to subjects with other types of dust exposure. Asbestos and silica/other dust-exposed subjects had better pulmonary function as compared to subjects with other types of dust exposure, but there were few significant findings.

In the subset of subjects with no documented previous dust exposure prior to their first PFT, the relatively poorer PFT performance of coal-exposed subjects as compared to subjects with other types of dust exposure was again evident, as was the relatively greater PFT performance of asbestos-exposed subjects.

4.5 X-Ray Analyses

4.5.1 Descriptive Statistics

Information regarding subject x-ray status as a function of exposure category is provided in Table 34. For the entire cohort (n= 29 237), just under half of subjects had one or more x-rays with an assigned ILO code. Among the three main dust exposure groups, the highest proportion of subjects with at least one coded x-ray were in the asbestos exposure group (60%), while the lowest proportion was in the coal group (37%). Among subjects with at least one x-ray with an assigned ILO code, the asbestos group had the highest proportion of subjects with pre-existing pneumoconiosis, at 1.7% (Table 34, part B). Because at least two x-rays are required to observe the development of incident pneumoconiosis, data was restricted to those subjects with more than one x-ray to obtain an estimate of incident pneumoconiosis (Table 34, part C). The proportion of subjects with incident pneumoconiosis was greatest for subjects with asbestos exposure (2.2%), and lowest for subjects with silica/other dust exposure (1.4%).

Table 35 provides similar information, but as a function of PFT group. Subjects with two or more PFTs were far more likely to have at least one ILO coded x-ray. This group also had the highest proportion of subjects with incident pneumoconiosis, at 2.5% (Table 44, part C). Although subjects with two or more PFTs only made up 41% (3728/9199) of subjects with two or more ILO coded x-rays, they represented 59% (95/162) of the incident pneumoconiosis cases. The greatest proportion of pre-existing pneumoconiosis (1.2% of subjects with x-ray data) was observed in the subjects without PFT data.

The 162 cases of incident pneumoconiosis and 145 cases of pre-existing pneumoconiosis were examined further. The frequencies of ILO codes are provided in Table 36. For subjects with incident pneumoconiosis, only 14 (8.6%) had a maximum ILO code for any of their coded x-rays that exceeded category 1. The corresponding number for subjects with pre-existing pneumoconiosis was 21 (14.5%).

From Table 36, it is also apparent that for a substantial number of subjects (34% of those with incident pneumoconiosis and 10% of those with pre-existing pneumoconiosis), by the time of their final x-ray their radiographs had "improved" to the point of no longer being positive for pneumoconiosis. With the exception of 1 subject with incident pneumoconiosis whose maximum ILO code was 2/2, all subjects who "improved" had a maximum ILO code in category 1.

Upon closer inspection of the assigned ILO codes for these subjects' x-rays, the vast majority had a single x-ray coded as 1/0 or 1/1, with previous and subsequent x-rays coded in category 0 (0/-, 0/0, or 0/1), and this likely represented intra- or inter-reader variability. In a few cases, the change from pneumoconiosis to normal occurred quite dramatically (ie a 5 to 6 point drop on the 12-point ILO scale) and over a short time period. For example, one subject had an ILO code of 2/2 in 1992, but his subsequent x-ray in 1994 was assigned the value 0/0. Another subject had an ILO code of 1/2 in 1992, but his subsequent x-ray in 1995 was coded as 0/-. Such changes in ILO coding likely reflected the change to the Hamilton, Ontario contract radiologist, who was tasked with assigning ILO codes after 1992. This is consistent with an earlier indication that the radiologist for the Fibrosis Program in the 1980's tended to assign higher ILO codes as compared to external readers in Ontario and the United States (Alleyne, 1988).

Figure 8 depicts the timing of when subjects with pneumoconiosis were identified. Subjects with incident pneumoconiosis were identified later on in the Fibrosis Program. Although weighted more towards the earlier years, subjects with pre-existing pneumoconiosis were identified throughout the span of the Fibrosis Program.

Subject age at the time of identification of pneumoconiosis is provided in Figure 9 (subjects with incident pneumoconiosis) and Figure 10 (subjects with pre-existing pneumoconiosis). Pneumoconiosis was identified before the age of 45 in 41 out of 162 subjects (25%) with incident pneumoconiosis and 56 out of 145 subjects (39%) with pre-existing pneumoconiosis.

Table 37 provides a summary of the duration of dust exposure prior to the identification of pneumoconiosis. For the 162 subjects with incident pneumoconiosis, 65 (40%) had an exposure duration under 10 years, and 32 (20%) had an exposure duration that was less than 5 years. Among the 145 subjects with pre-existing pneumoconiosis, only 53 had a documented occupational exposure history at entry into the Fibrosis Program. Of these 53 subjects, all but one had an explicitly documented previous occupational exposure history of zero years at the time of their entry into the Fibrosis Program.

4.5.2 Pneumoconiosis and Pulmonary Function

Table 38 provides a comparison of mean PFT indices for the first PFT across x-ray status groups for all subjects with at least one PFT (n = 7763). Subjects with normal x-rays had the highest mean FEV_1 and FVC values, followed by subjects with no x-ray data, subjects with incident pneumoconiosis, and then subjects with pre-existing pneumoconiosis. The absolute mean difference in FEV_1 and FVC between subjects with normal x-rays and subjects with incident pneumoconiosis was slightly greater than 600 mL. Subjects without x-ray data had a higher FEV_1/FVC% than all other x-ray status groups.

The subset of subjects with height data was evaluated using percent-predicted values. With the exception of subjects with normal x-rays, subject numbers in the other x-ray status categories were quite small. Despite low subject numbers, those with incident pneumoconiosis had significantly lower percent-predicted FEV_1 and FVC as compared to subjects with normal x-rays.

A similar comparison for the subjects with two or more PFTs (n = 4206) is provided in Table 39. Similarly to the whole PFT cohort, subjects with normal x-rays had the highest mean FEV_1 and FVC at both the first and last PFT. The absolute mean difference in FEV_1 and FVC between subjects with normal x-rays and subjects with pre-existing pneumoconiosis (the x-ray status group with the lowest mean values) was slightly more than 900 mL at the first PFT and 1100 mL at the last PFT. Absolute mean differences in FEV_1 and FVC between subjects with normal x-rays and subjects with incident pneumoconiosis were slightly more modest, at just under 600 mL at the first PFT and just under 700 mL at the last PFT. For the subset of subjects with height data, subjects with incident pneumoconiosis had consistently lower percent-predicted FEV_1 and FVC at both the first and last PFT, although this was only significant for percent-predicted FEV_1 at the first PFT. Subject numbers were quite low in this subset.

Table 40 compares mean ages, PFT dates, and duration between the first and last PFT across x-ray status groups. Both subjects with incident pneumoconiosis and subjects with pre-existing pneumoconiosis were considerably older than subjects with normal x-rays. For the subset of subjects with two or more PFTs who had data on height (n = 2421), there were no significant differences in height across x-ray status groups.

4.5.3 Pneumoconiosis and Pulmonary Function, Regression Analyses

Regression analyses focused on the subset of subjects with two or more PFTs. Subjects with incident (n = 95) and pre-existing (n = 29) pneumoconiosis were combined into a single group for comparison with subjects with normal x-rays (n = 3612). Subjects with no x-ray data were excluded from all regression analyses. Included in the pneumoconiosis group were 66 subjects with coal exposure (54 incident, 12 pre-existing), 30 subjects with silica/other exposure (22 incident, 8 pre-existing), and 28 subjects with asbestos exposure (19 incident, 9 pre-existing). Included in the subjects with normal x-rays group were 1927 subjects with coal exposure, 1025 subjects with silica/other dust exposure, 638 subjects with asbestos exposure, and 22 subjects with MMMF exposure.

Simple linear regression using pneumoconiosis (presence or absence) as a single variable indicated that subjects with pneumoconiosis had a mean FEV_1, FVC, and FEV_1/FVC% that was 660 mL, 659 mL, and 2.82% lower, respectively, than subjects with normal x-rays at the first PFT. At the last PFT, subjects with pneumoconiosis had a mean FEV_1, FVC, and FEV_1/FVC% that was 777 mL, 791 mL, and 3.89% lower, respectively, than subjects with normal x-rays. All pneumoconiosis regression coefficients were significant at the $p < 0.001$ level.

First PFT outcomes were then assessed using a multiple linear regression that included pneumoconiosis, age, sex, and PFT date as variables. At the first PFT, subjects with pneumoconiosis had a mean FEV_1 and FVC that were 257 mL and 239 mL lower, respectively, than subjects with normal x-rays ($p < 0.01$). The coefficient for FEV_1/FVC% indicated a 1.43% lower value for subjects with pneumoconiosis, but this was not significant ($p = 0.059$).

This regression analysis was then repeated on the subset of subjects with height data ($n = 2418$) with height included as an additional variable in the regression. There were 2361 subjects with normal x-rays (1365 coal, 654 silica/other dust, 327 asbestos, 15 MMMF) and among subjects with pneumoconiosis ($n = 57$), there were 37 subjects with coal exposure (33 incident, 4 pre-existing), 11 subjects with silica/other exposure (9 incident, 2 pre-existing), and 9 subjects with asbestos exposure (7 incident, 2 pre-existing). Subjects with pneumoconiosis had a mean FEV_1, FVC, and FEV_1/FVC% that were 128 mL, 84 mL, and 1.140% lower, respectively, than subjects with normal x-rays, but none of the pneumoconiosis coefficients reached significance ($p = 0.14$, $p = 0.38$, $p = 0.27$, respectively).

Multiple linear regression analyses were then performed for the last PFT, which included age, sex, date of PFT, duration between the first and last PFT, corresponding first PFT value, and pneumoconiosis as variables. Subject numbers were the same as for the previous regressions, above. The coefficients for pneumoconiosis were again all negative, indicating lower values in subjects with pneumoconiosis as compared to subjects with normal x-rays. The values were –158mL for FEV_1 ($p = 0.001$), -142 mL for FVC ($p = 0.012$), - 1.481% for FEV_1/FVC% ($p = 0.015$).

Similarly to the multiple linear regression analyses for the first PFT on the subset of subjects with height data, including height as a variable attenuated the pneumoconiosis coefficients for the last PFT as well. In these regression analyses, subjects with pneumoconiosis had a mean FEV_1, FVC, and FEV_1/FVC% that were 74 mL, 21 mL, and 1.825% lower, respectively, than subjects with normal x-rays, but only the pneumoconiosis coefficient in the FEV_1/FVC% analysis reached significance ($p = 0.26$, $p = 0.78$, $p = 0.03$, respectively).

4.6 Smoking Analyses

4.6.1 Descriptive Comparisons

As indicated in Table 4, very few subjects in the Fibrosis Program database had any smoking documentation: only 732 (2.5%) subjects out of the entire cohort of 29 237. Subjects with PFT data were more likely to have smoking information than subjects without PFT data, but the overall numbers were still quite low: 647 / 7763 (8.3%) for subjects with at least one PFT, and 321 / 4206 (7.6%) for subjects with two or more PFTs. The breakdown of subject numbers by smoking status is provided in Table 41.

Because of low subject numbers, only the dichotomous category of ever smokers versus never smokers was used for PFT comparisons within the different dust exposure groups. Table 42 summarizes mean pulmonary function parameters for subjects with at least one PFT. There were no significant differences in pulmonary function parameters between ever smokers and never smokers for coal-exposed subjects. For silica/other dust-exposed subjects, FVC and percent-predicted FVC were significantly greater and FEV_1/FVC% was significantly smaller for ever smokers as compared to never smokers. For asbestos-exposed subjects, the only significant pulmonary function mean difference was a 467 mL lower FEV_1 in ever smokers.

Table 43 summarizes mean pulmonary function parameters for subjects with two or more PFTs. There were no significant differences in pulmonary function parameters between ever smokers and never smokers for coal-exposed subjects. With the exception of a 4.5% greater FEV_1/FVC% in never smokers at the first PFT, there were no significant mean differences between ever and never smokers for silica/other dust-exposed subjects. For asbestos-exposed subjects, never smokers had a significantly greater FEV_1 and FVC at the first PFT and a significantly greater FEV_1 at the last PFT as compared to ever smokers.

4.6.2 Regression Analyses, First PFT

The influence of smoking on pulmonary function parameters for the subset of workers with smoking data was also explored in regression analyses, focused on within exposure group assessments. For the first PFT, simple linear regression analyses are summarized in Table 44. Smoking had the strongest effect on FEV_1 for the 100 asbestos-exposed subjects: never smokers had a 439 mL greater FEV_1 than ever smokers. The greatest effect on FEV_1/FVC% was observed in silica/other dust-exposed subjects: never smokers had an almost 5% greater value than ever smokers.

The subjects with smoking data were then analyzed using multiple linear regressions with smoking included (Table 45) and excluded as a variable in the regression model (Table 46), for first PFT parameters. The only significant relationships with smoking were observed in the silica/other dust exposure group: never smokers had an FVC that was 345 mL lower than ever smokers; $FEV_1/FVC\%$ was 4.7% greater in never smokers. Regardless of presence or absence of smoking in the regression model, previous dust exposure was not a significant predictor of pulmonary function, with the exception of FVC for coal-exposed workers. With smoking excluded from the regression model, the effect of dust exposure on FVC for coal-exposed subjects was minimally affected (-20 mL per year of previous exposure when smoking included versus –21 mL per year of previous exposure when smoking was excluded).

4.6.3 Regression Analyses, Last PFT

Subject numbers dropped substantially for analyses that focused on last PFT parameters, and only 303 subjects (two thirds of whom were coal-exposed subjects) in total were included in analyses. There was no significant effect of smoking on PFT parameters in either simple linear regression (Table 47) or multiple linear regression analyses (Table 48). There were no significant effects of dust exposure duration on pulmonary function at the last PFT for this subset of subjects, regardless if smoking was included (Table 48) or excluded (Table 49) in the regression model.

5.0 Discussion

5.1 Main Findings, Within Exposure Group Analyses

Roughly one quarter of subjects had dust exposure prior to their first PFT. Therefore, the first Fibrosis Program PFT was not a pre-exposure "baseline" measurement. For coal and asbestos-exposed subjects, however, when dust exposure prior to the first PFT was examined as a continuous variable there were no significant effects on pulmonary function. A different picture emerged for silica/other dust-exposed subjects, who experienced a 9 mL loss in FEV_1 and 0.19% loss in percent-predicted FEV_1 for every year of previous dust exposure, based on multiple linear regression models that included other key variables (Tables 10 and 11). This observed loss in FEV_1 as a function of duration of exposure is consistent with the 100 to 300 mL loss of FEV_1 over a working career attributable to silica exposure that has been previously described (Cowie et al., 1991; Hertzberg et al., 2002; Hnizdo, 1992).

When exposure that occurred during enrolment in the Fibrosis Program was also considered, the deleterious relationship between dust exposure and pulmonary function for silica/other dust-exposed subjects was no longer observed at the time of the last PFT. The only significant finding from multiple linear regression analyses for these subjects was a 0.151% increase in percent-predicted FEV_1 for each year of dust exposure. When subjects who only had exposure during enrolment in the Fibrosis Program were considered, each year of dust exposure was associated with a 0.552% increase in percent-predicted FEV_1.

In contrast to the lack of an observed relationship between previous dust exposure and first PFT outcomes for coal and asbestos-exposed subjects, each year of dust exposure prior to the last PFT was associated with a 9 mL increase in FVC for coal-exposed subjects and a 10 mL decrease in FEV_1 for asbestos-exposed subjects when all subjects with two or more PFTs were considered (Table 20). For subjects with ten or more years exposure duration, coal-exposed subjects experienced a 15 mL and 17 mL increase in FEV_1 and FVC, respectively, and asbestos-exposed subjects experienced a 12 ml and 9 mL decrease in FEV_1 and FVC, respectively (Table 29). The observations for asbestos-exposed subjects are similar to those of Siracusa et al. (1984), who noted a 16 mL decline in FEV_1 and FVC per year of asbestos exposure. The positive effect of dust exposure for coal-exposed subjects and negative effect of dust exposure for asbestos-exposed subjects was also consistently observed for percent-predicted FEV_1 and FVC, and for the subset of subjects with no dust exposure prior to the first PFT.

Based on within exposure group analyses, exposure prior to the first PFT only seemed to affect silica/other dust-exposed subjects. However, exposure prior to the last PFT, including exposures experienced prior to and during enrolment in the Fibrosis Program, had no significant effect on pulmonary function of silica/other dust-exposed subjects, a significant positive effect for coal-exposed subjects, and a significantly negative effect on asbestos-exposed subjects.

5.2 Main Findings, Between Exposure Group Analyses

The aim of this study was to examine longitudinal changes in pulmonary function for subjects enrolled in the Alberta Fibrosis Program, and it was hypothesized that pulmonary function parameters at last contact with the Program would not differ significantly between workers exposed to coal, silica, or asbestos dusts. For silica/other dust and asbestos-exposed subjects, this hypothesis was essentially valid. However, despite the positive relationship between dust exposure and pulmonary function for coal-exposed subjects, by the time of the last PFT, these subjects had a 57 mL lower FEV_1 and 82 mL lower FVC as compared to subjects with other types of dust exposure (Table 22). When subjects with ten or more years exposure were considered, these values were 69 mL lower and 84 mL lower, respectively (Table 31). The poorer PFT performance of coal-exposed subjects was consistently observed for percent-predicted FEV_1 and FVC, and for the subset of subjects with no dust exposure prior to the first PFT.

The interpretation of this observation is unclear for a number of reasons. Firstly, subjects in the three dust-exposure groups demonstrated significant differences in pulmonary function at their first "baseline" PFT. The FEV_1 and FVC of coal-exposed subjects were not significantly different from the corresponding indices for subjects exposed to other dust types at the first PFT. However, subjects with silica/other dust exposure had a 69 mL lower FEV_1 and 61 mL lower FVC at the first PFT, while asbestos-exposed subjects had 152 mL greater FEV_1 and 65 mL greater FVC (Table 12). The fact that the asbestos-exposed subjects did not maintain their better performance over other dust types by the time of their last PFT could imply that these subjects experienced greater pulmonary function losses during their enrolment in the Fibrosis Program than coal or silica/other dust-exposed subjects.

Secondly, the interpretation of the results is limited due to the nature of the internal comparisons between subjects who were all dust-exposed. The design of this study could only detect relative differences in pulmonary function between different dust exposure groups; a finding of "no significant differences between groups" is moot if the three dust exposure groups have experienced similar clinically significant absolute losses in pulmonary function. The ideal would be to compare each dust exposure group with a suitably comparable group of unexposed referents. This was not possible given the design of the Fibrosis Program, as it was strictly a surveillance program for exposed workers.

Without a well-matched unexposed comparison group, the only way to place the observed pulmonary function values of exposed workers into context is to rely on prediction equations derived from general population studies. The prediction equations of Morris et al. (1971) were used to derive percent-predicted FEV_1 and

FVC values for dust-exposed subjects in the present study. More recent general population spirometry reference values have been published (Hankinson et al., 1999), based on data from the third National Health and Nutrition Examination Survey (NHANES III), and the use of these reference values has been recommended by the American Thoracic Society and the American College of Occupational and Environmental Medicine (Townsend et al., 2000; Weir et al., 2005). Nevertheless, the Morris et al. (1971) prediction equations were used, as they were the ones that were specifically recommended by the Fibrosis Program (Fibrosis Program Information Manual, 1981). It has been noted, however, that the Morris et al. (1971) norms were based on a sample of an American population and therefore may not have been representative of the working Alberta population (Fibrosis Program Information Manual, 1981).

Based on these prediction equations, the mean percent predicted FEV_1 and FVC at the time of the last PFT were well within normal limits for all dust exposure groups, ranging from 97% to 103% (Tables 24 and 33) suggesting that any mean relative differences between dust exposure groups were of no clinical significance. However, the restriction of the analysis to subjects with at least two PFTs likely produced a positive bias: workers that may have experienced adverse effects due to dust exposure and left the industry would have been lost to follow-up, resulting in higher mean lung function levels than if all workers had been included in the analysis. This bias would be age-related as well, and result in reduced estimates of rate of loss of lung function when compared to predicted lung function values obtained from cross-sectional population surveys (Vollmer et al., 1993).

This probable healthy worker bias is reflected in the apparent "improvement" in mean percent-predicted FEV_1 and FVC at the last PFT as compared to the first PFT. For example, mean percent predicted FEV_1 for subjects with ten or more years exposure duration improved by roughly 6.4%, 7.0%, and 4.6% for coal, silica/other dust, and asbestos-exposed subjects, respectively. Simply comparing mean percent predicted values could suggest that the asbestos-exposed subjects faired the poorest of the dust exposure groups, as they had the smallest degree of "improvement" in percent-predicted FEV_1 by the time of the last PFT. However, this interpretation is difficult to reconcile with the observation that the asbestos-exposed subjects had the greatest percent-predicted FEV_1 values of all dust exposure groups.

The limitations inherent in comparing workplace periodic PFT screening results with population reference values have been noted by the American College of Occupational and Environmental Medicine. Because of their health, workers often have lung function that is greater than 100% of the predicted value. Subsequently, these workers may lose large fractions of their lung function and still be within the "normal" range and so comparison to predicted values may not detect serious pulmonary function deterioration (Townsend et al., 2005). Based on overall findings, it would appear that subjects in the Fibrosis Program with two or more PFTs did not experience any clinically significant decrements in pulmonary function due to dust exposure. However, others have cautioned that substantial negative effects of dust exposure in a small proportion of susceptible individuals may not be readily apparent if the majority of observed subjects experience little to no adverse effects of dust exposure on pulmonary function (Becklake, 1985; Stenton et al., 1993). Indeed, declines in pulmonary function due to dust exposure can be considerable in some susceptible individuals (Beeckman et al., 2001; Hurley et al., 1986; Soutar et al., 1986; Soutar et al., 1993; Soutar et al., 2004).

Although the effects of dust exposure on pulmonary function were apparently not substantial in this study, this is not necessarily unexpected given that few subjects were monitored for a sufficient period of time for the adverse effects of dust exposure to manifest. Coal workers' pneumoconiosis, silicosis, and asbestosis are chronic lung diseases that require many years, if not decades, of cumulative dust exposure to develop (Banks et al., 2005; Brodkin et al., 2005; Mossman et al., 1998; Petsonk et al., 2005). For example, in the Ontario silicosis surveillance program, less than 2 new cases of silicosis were identified per 10000 examinations within twenty years of first exposure, but this increased to 20-40 new cases per 10000 examinations when the time from first exposure was more than 27 years (Finkelstein, 1995). In their study of asbestos-cement workers, Siracusa et al. (1988) observed that annual declines in lung function were close to expected age-related declines for subjects with less than 15 years of dust exposure, whereas annual declines in FEV_1 and FVC were more than 50 mL for subjects with more than 15 years of dust exposure. U.S. surveillance has consistently demonstrated that CWP is rarely observed in coal miners with less than 10 years tenure (CDC, 2003; NIOSH, 2003). The fact that the silicosis exposure-response risk estimates obtained by Muir et al. (1989b) were lower than other studies was attributed to a shorter follow-up period (Hnizdo et al., 1993; Steenland et al., 1995).

In the present study, 2130 out of 7763 subjects (27%) with any PFT data had at least ten years of dust exposure prior to either their first or last PFT. Since subjects who may have suffered adverse effects due to dust exposure and left the industry would have been lost to follow-up, this likely would have led to a survivor bias in the analysis of subjects who had longer periods of time between their first and last PFTs. Unless the "drop-out" subjects were included, analysis only of survivors likely would have underestimated the adverse affects of dust exposure (Eisen et al., 1995). In the present study, silica/other dust and asbestos-exposed subjects with ten or more years exposure duration had similar mean last PFT values as subjects with less than ten years exposure duration. Percent-predicted FEV_1 and FVC values at the last PFT were also virtually identical for these two groups of subjects, despite a difference in mean duration of exposure of almost 12-13 years. This is either suggestive of no adverse effect of dust exposure or some degree of survivor bias, but without pulmonary function data on subjects lost to follow-up, the distinction between the two explanations cannot be addressed definitively.

5.3 Main Findings - X-ray Analyses

A total of 307 subjects with radiographic pneumoconiosis (ILO profusion score ≥ 1/0) were identified out of 14239 subjects with at least one ILO coded x-ray, for an overall crude prevalence of 2.1%. Of these subjects, almost half had pneumoconiosis at their first x-ray (pre-existing pneumoconiosis). For subjects with at least two x-rays (n = 9199), 1.8% who originally had a normal x-ray developed radiographic pneumoconiosis during their enrolment in the Fibrosis Program. However, An earlier x-ray validation study demonstrated that of 70 x-rays graded 1/1 or greater by the Fibrosis Program radiologist, only 11 were graded similarly by external readers in the United States and Ontario, suggesting a high likelihood of false positive radiographic pneumoconiosis readings (Alleyne, 1988). It was also noteworthy that 34% of subjects who developed radiographic pneumoconiosis at some point during their enrolment in the Program had "improved" to within the normal range by the time of their last x-ray. For several subjects with unequivocal pneumoconiosis (ILO profusion ≥ 1/1), their profusion score improved dramatically within a period of two to three years, and this likely reflected the change to the Hamilton, Ontario contract radiologist, who was tasked with assigning ILO codes after 1992. Although difficult to assess definitively without a further validation study, it is highly likely that the present x-ray data overestimated the true prevalence of radiographic pneumoconiosis in the study population.

For coal-exposed subjects, when both pre-existing and incident pneumoconiosis were combined, the crude prevalence of radiographic CWP ranged from 2.2% for subjects with at least one ILO coded x-ray to 2.8% for subjects with more than one ILO coded x-ray. Using data collected from just over 32000 miners from 1995 to 2002, the United States' Coal Workers' X-ray Surveillance Program found that the crude prevalence of CWP was 2.8% (CDC, 2003). In the United Kingdom, a recent survey of 15 collieries observed a 0.8% prevalence of CWP from 1998-2000 (Scarisbrick et al., 2002). Of course, it is important to consider that the overall prevalence of CWP in both of these countries has been declining steadily since the early 1970's, and that these prevalence estimates are from more modern times; the reported crude prevalences of CWP in the late 1960's and into the 1970's were greater than 10% in the U.S. and U.K. (Attfield et al., 1992; CDC, 2003; Goodwin et al., 1998; NIOSH, 2003; Scarisbrick et al., 2002). Given that the Fibrosis Program data extended back to 1964 and that the crude CWP prevalence for the whole Program was comparable to modern times, it would appear that the overall burden of CWP in Alberta workers was lower than in the U.S. and U.K. This would not be entirely unexpected, since the U.S. and U.K. surveys were focussed on underground coal-miners. The predominant type of coal mining in Alberta has been surface mining (Kaegi, 1981), and although few studies have been performed on surface miners, available data suggest that their coal dust exposures are less, and there is little impact of surface coal mining on pulmonary function or radiographic abnormalities (Love et al., 1997).

For silica/other dust-exposed workers, when both pre-existing and incident pneumoconiosis were combined, the crude prevalence of radiographic silicosis ranged from 1.9% for subjects with at least one ILO coded x-ray to 2.3% for subjects with more than one ILO coded x-ray. In their study of Ontario hard rock miners, Muir and colleagues identified 32 silicosis cases out of 2109 subjects, for a crude prevalence of 1.5% (Muir et al., 1989b). Their cut-off for radiographic pneumoconiosis was an ILO profusion score of 1/1 or greater; in the present analysis, 46 silica/other dust-exposed subjects had an equivalent ILO profusion, or 0.9% of subjects with at least one ILO coded x-ray. An Ontario surveillance program identified 283 silicosis cases out of 68701 workers (0.4%) (Finkelstein et al., 1994). Therefore, the crude prevalence of silicosis in the Alberta Fibrosis Program is on par with Ontario studies (if one ignores the high likelihood that the Fibrosis Program overestimated the number of radiographic pneumoconiosis cases).

For asbestos-exposed workers, when both pre-existing and incident pneumoconiosis were combined, the crude prevalence of radiographic asbestosis ranged from 2.9% for subjects with at least one ILO coded x-ray to 3.9% for subjects with more than one ILO coded x-ray. There are limited studies with which to compare these figures. Most large surveys of asbestos-exposed workers have focussed on tradespeople with a long duration of exposure. This restriction, coupled with the inclusion of both active and retired workers would likely result in larger prevalence estimates for a long latency disease such as asbestosis than if only active workers were included, regardless of tenure. In these surveys, asbestosis prevalence ranged from 12% to 60% (Miller et al., 1992; Selikoff et al., 1991; Welch et al., 1994). In Canada, the prevalence of asbestosis was 6.9% among employees of the asbestos mining industry in Quebec in 1976 (Ostiguy et al., 1979).

A dose-response relationship exists between cumulative dust exposure and pneumoconiosis and it has been consistently observed that subjects with radiographic pneumoconiosis have lower pulmonary function than dust-exposed subjects with normal chest radiographs (Gamble et al., 2004; Hnizdo, 1992; Irwig et al., 1989; Kilburn et al., 1994; Miller et al., 1992; Miller et al., 1996; Rogan et al., 1973; Rosenstock et al., 1988). In the present analysis, subjects with radiographic pneumoconiosis had consistently lower pulmonary function than subjects with normal radiographs, which was consistent with earlier studies. At the time of the first PFT, the combined group of subjects with pre-existing and incident pneumoconiosis had an FEV_1 and FVC that were more than 600 mL lower than subjects with normal x-rays, and this difference increased in magnitude by over 100 mL by the time of the last PFT. These differences were attenuated considerably in multiple linear regression analyses; although the negative relationship between pneumoconiosis and pulmonary function remained, differences were no longer statistically significant.

If there was no dust exposure prior to the first PFT, it would not be expected that subjects who would go on to develop radiographic pneumoconiosis would have poorer pulmonary function at baseline. Of the 57 subjects with pneumoconiosis included in the multiple linear regression analysis that incorporated height as an independent variable, 49 (86%) had normal x-rays at entry into the Fibrosis Program. Of these, 30 had zero years of previous dust exposure at the time of their first PFT. The multiple linear regression analysis was repeated with only these 30 subjects with incident pneumoconiosis plus the subjects with normal x-rays. The resultant non-significant pneumoconiosis coefficients for FEV_1, FVC, and $FEV_1/FVC\%$ were - 150 mL, - 72 mL, and - 1.5%, respectively, which were very similar to the results obtained from the original analysis that included pre-existing pneumoconiosis cases and incident pneumoconiosis cases with previous dust exposure (- 128 mL, - 84 mL, and – 1.1%, respectively, all not significant). Although none of these pneumoconiosis coefficients reached statistical significance, the similarity of the results for the 30 incident pneumoconiosis subjects with no history of previous dust exposure suggests that the assessment of previous dust exposure was likely inadequate (discussed further in section 5.4.2).

5.4 Study Limitations

The overall results of this study must be interpreted with caution, owing to the numerous limitations in the data and the analysis. It is important to emphasize that the Alberta Fibrosis Program was designed as a surveillance tool; epidemiological study was not necessarily the intent for the collected data. These limitations can be broadly divided under the categories of subject selection, exposure, confounding, outcome, and analysis.

5.4.1 Selection

All surveillance programs are subject to potential selection bias as only workers who are healthy enough to work will be included in such a program; workers who become ill or disabled due to dust exposure and leave their dusty jobs will not be recognized under a surveillance program, which may underestimate the true burden of disease (Attfield et al., 1992a; NIOSH et al., 2003). In studies that have included ex-workers, for example, more abnormalities were observed in the ex-workers than actively employed workers (Eisen et al., 1995; Naidoo et al., 2005; Oxman et al., 1993; Vollmer et al., 1993). Pneumoconiosis is a chronic disease with a long latency after first exposure, and so surveys of only active workers will not identify disease that develops in workers after retirement (Malmberg et al., 1993; Soutar et al., 1986). The long latency of pneumoconiosis also means that surveys that are heavily weighted with young workers who are initially surveyed and then lost to follow-up or who do not stay in the industry for very long will potentially "dilute" the prevalence of chronic health effects that may only be observed in the minority of workers who are surveyed over a long period of time.

It is highly likely that the Alberta Fibrosis Program suffered from a substantial degree of selection bias. Firstly, according to regulations pertaining to the Program, the employer was responsible for identifying exposed workers and (after 1971) paying for all biennial examinations. Because of this "self-identification" it is quite plausible that employers with a strong sense of due diligence with respect to worker health and safety would have been more inclined to participate in the Fibrosis Program than employers with more lax attitudes towards exposure controls and sound industrial hygiene practice. Workers at greater risk for experiencing adverse pulmonary effects due to dust exposure may therefore have been underrepresented in the Program. Unfortunately, there is inadequate data to judge if this was indeed the case, simply because it was never verified if inclusion criteria were followed. In the United States, only 30% of underground coal miners participate in the NIOSH-administered Coal Workers' X-ray Surveillance Program (NIOSH, 2003). Whether or not this participation rate is reflective of Alberta is uncertain, given that "an effective means for identifying every company where there may be workers exposed to asbestos, silica, or coal dust is not available and a reliable estimate of the number of Alberta workers who should be covered by the Fibrosis Program is not possible" (Alleyne, 1988). There is some evidence to suggest that participation in the Fibrosis Program was poor: in an analysis of 36 asbestosis claims accepted by the Alberta Workers' Compensation Board, only four were found to be workers who had participated and had records in the Fibrosis Program (Alleyne et al., 1994).

Secondly, there are indications that many workers who underwent initial examinations in the Fibrosis Program were lost to follow-up. Kaegi (1981) noted that among coal mining companies, comprehensive examinations were conducted at the time of hiring, as required, but periodic medical assessments thereafter were "infrequent, incomplete, or non-existent". Based on submitted x-ray data, only 34.5% of expected second x-rays were submitted, dropping steadily to only 2.3% of expected sixth x-rays (Alleyne, 1982). The present analysis also suggests that the Fibrosis database was weighted towards "one-time" evaluations: of the 7763 subjects with PFT data, only 4206 (54%) subjects had two or more PFTs; of the 14239 subjects with at least one x-ray with an assigned ILO code, 5040 (35%) had only a single ILO-coded x-ray.

Thirdly, it is not entirely clear why only 14% of subjects (4206 out of 29237) had adequate data to warrant a longitudinal analysis. For example, were these simply workers of diligent employers who ensured strict adherence to provincial occupational health and safety regulations? If this were true, then it is likely that workplaces with poor regulation compliance (and therefore potentially lax dust exposure controls) were underrepresented in the analysis. This would potentially underestimate any adverse dust effects. Alternatively, these workers may have been identified as "at risk" of developing pneumoconiosis, based on current exposures, historical exposures, or clinical suspicion, and subsequently were placed under more thorough surveillance.

Perhaps this would partly explain why at the time of the first PFT, workers with two or more PFTs had significantly lower percent-predicted FEV_1 and FVC as compared to workers with less than two PFTs (Table 15). As noted in Table 27, subjects with ten or more years exposure duration had significantly lower percent-predicted FEV_1 and FVC values at the first PFT. However, when only those subjects with no previous exposure prior to the first PFT were examined, significantly lower percent-predicted FEV_1 and FVC values for subjects who would eventually go on to have ten or more years exposure duration were still observed, ranging from 13% lower for coal-exposed subjects to 4% lower for silica/other dust and asbestos-exposed subjects. As well, among workers with more than one x-ray (Table 35, part C), subjects with two or more PFTs had a higher proportion of subjects with incident radiographic pneumoconiosis as compared to other subjects. However, one would suspect that the identification of radiographic pneumoconiosis at the time of the first x-ray would also signal the need for closer surveillance, and yet workers with two or more PFTs had the lowest prevalence of pre-existing pneumoconiosis (Table 35, Part C) as compared to other subjects.

Unfortunately, it is not possible to determine the proportion of workers at risk for pneumoconiosis in Alberta who were included in the Fibrosis Program, nor can it be determined from available data why only a small proportion of subjects in the Fibrosis Program database had adequate data for longitudinal pulmonary function analyses. Subsequently, the magnitude and direction of any potential selection bias is unclear.

5.4.2 Exposure

Another major limitation of the Fibrosis Program database was the assessment of exposure. Originally, workers were to be included in the Program if they belonged to a particular occupation (Table 1). It was not until the early 1980's that exposure criteria for the inclusion of workers in the Fibrosis Program were stipulated (Table 2), but it was not verified if inclusion criteria were followed (Alleyne, 1982; Alleyne, 1988). Workers exposed to the same dust type but in different occupations, or workers in the same occupation but working for different companies, may have had vastly different dust exposures in terms of intensity, frequency, and cumulative dose, but they would all be included in the same dust exposure category. Because exposure was simply scored as "exposed" or "unexposed", it is impossible to determine the degree of exposure for any individual worker. Subsequently, an adverse effect on pulmonary function that is only observed at high exposure levels may be "diluted" if a small number of workers with high exposures are included in the same group as workers with low exposure for analytical purposes.

It is impossible to confirm if the present analysis adequately identified all subjects with previous dust exposure prior to their first PFT. However, based on the analyses of radiographic pneumoconiosis, there is a strong indication that previous occupational exposure history was poorly documented in at least some cases. For example, virtually all subjects with pre-existing pneumoconiosis who had a documented previous occupational exposure history had a duration of previous exposure that was zero years (Table 37). As well, one fifth of subjects with incident pneumoconiosis had an exposure duration that was less than five years, which is not consistent with the typically long latency that is normally observed from the onset of dust exposure to the identification of radiographic pneumoconiosis. If previous occupational exposure was underestimated in a sizable proportion of this subset of subjects, then it is likely that it was also underestimated for other subjects as well.

The present analyses suggest that negative effects of dust exposure prior to the first PFT were evident only for silica/other dust-exposed subjects. However, a different picture emerged when exposures that occurred during the Fibrosis Program were also considered in the examination of the effects of dust exposure on the last PFT (see Section 5.1). These discrepant results may be related to the probable inadequacy of the assessment of previous dust exposure (as evidenced by the evaluation of radiographic pneumoconiosis, discussed above). Therefore, it is difficult to make firm conclusions regarding the potential impact of dust exposures that occurred prior to enrolment in the Fibrosis Program.

Underestimation of previous dust exposure could have had implications for the observed effects of dust exposure on last PFT parameters. For example, several studies have suggested that pulmonary function deficits associated with coal dust are greatest during the initial years of exposure, with continued dust exposure resulting in relatively smaller declines thereafter (Carta et al., 1996; Henneberger et al., 1996; Henneberger et al., 1997; Seixas et al., 1992; Seixas et al., 1993; Wang et al., 2005). If a substantial proportion of subjects in the present study had dust exposure prior to their first PFT, then the "first" PFT would have potentially recorded "baseline" pulmonary function after an initial period of dust-related pulmonary function decline. Subsequent observed dust-related changes between the first and last PFT, if any, would therefore underestimate the true dust exposure effect.

Whether or not this potential underestimation of the influence of dust exposure on pulmonary function affected the overall conclusions of the present analysis is difficult to determine with certainty. However, mean percent predicted FEV_1 and FVC at the time of the last PFT ranged between 97% and 103% across exposure groups (Tables 24 and 33). Therefore, on average, these subjects were still well within the range of normal pulmonary function at the time they left the Fibrosis Program.

5.4.3 Confounding

The interpretation of comparisons between different dust exposure groups is limited due to the inability to control for several potential confounding variables.

The exposure code used in the Fibrosis Program did not capture whether or not subjects were exposed to other substances at work that could also impact on pulmonary function, such as diesel exhaust (Bakke et al., 2004; Sydbom et al., 2001), welding fumes (Erkinjuntti-Pekkanen et al., 1999; Ozdemir et al., 1995), or other dusts. Additionally, there is no information in the Fibrosis Program database on the use of respiratory protective equipment. The use of respirators in dusty occupations has been shown to be protective against the adverse pulmonary effects of dust exposure (Li et al., 2002; Wang et al., 1999a). Systematic differences between dust exposure groups in the present study may have been responsible for some of the observed differences. For example, if coal miners had considerable exposure to diesel exhaust and were less inclined to use a respirator than silica-exposed subjects, then lower pulmonary function in coal-exposed subjects may not have been completely explained solely by the difference in type of dust exposure.

Height is a significant predictor of pulmonary function indices such as FEV_1 and FVC (ATS, 1991). Unfortunately, height data was only available for 47% to 65% of subjects with PFT data, depending on the analysis. Separate multiple linear regression analyses (data not shown) were also performed that examined all subjects with PFT data and then separately for the subset of subjects with height data, but with height not included in the regression model. In other words, the same independent variables were included in these regression models. When between exposure group analyses were performed, exposure type coefficients varied considerably between these two sets of multiple linear regressions, suggesting that results obtained from the subset of subjects with height data may not be completely representative of all subjects with PFT data. It is unclear how the results may have differed, if at all, if all subjects with PFT data could have been included in analyses.

The Fibrosis Program did not collect data on subject weight. Several studies have determined that body weight, typically measured as Body Mass Index (BMI), is significantly related to pulmonary function, and that weight gain is associated with longitudinal pulmonary function decline (McKay et al., 1999; Morgan et al., 2000; Wang et al., 1997b). The potential confounding influence of systematic differences in subject body weight between different dust exposure groups cannot be assessed with the available data.

The Fibrosis Program also did not collect data on subject ethnicity, which is known to affect measured pulmonary function. For example, Hankinson et al. (1999) noted African-Americans have lower FEV_1 values for a given height as compared to Caucasians. This was attributed to differences in body build, in that

African-Americans, on average, have a smaller trunk to leg ratio than Caucasians. Indeed, Hankinson et al. (1999) provide distinct spirometric reference value prediction equations for three different ethnic groups: Caucasian, African-American, and Mexican-Americans. The potential confounding influence of systematic differences in subject ethnicity between different dust exposure groups cannot be assessed within the available data, although it seems unlikely that there would have been systematic differences in ethnicity between exposure groups.

Perhaps the most important potential confounder, smoking, could not be accounted for in the main analyses of pulmonary function. The literature on the three dust types is consistent with the conclusion that the combined effects of smoking and dust exposure on pulmonary function are greater than dust exposure alone, and in some cases, decrements in pulmonary function of any clinical significance in dust-exposed workers are only observed in smokers (Alfonso et al., 2004; Algranti et al., 2001; Attfield et al., 1992; Chia et al., 1992; Erdinc et al., 2003; Hnizdo et al., 1990; Kilburn et al., 1994; Kriess et al., 1989; Love et al., 1982; Malmberg et al., 1982; Marine et al., 1988; Miller et al., 1992; Ohlson et al., 1984; Siracusa et al., 1984; Soutar et al., 1986; Weill et al., 1975; Wiles et al., 1977).

Smoking data was only available on a small subset of subjects. There were few significant observed relationships between smoking and pulmonary function. As well, the presence or absence of smoking as a variable in the multiple linear regression models had no effect on the relationship between dust exposure and pulmonary function. Nevertheless, subject numbers in the smoking comparisons were small, and the representativeness of the results for the larger group of subjects with PFTs is unclear. Few conclusions can be drawn from these analyses with respect to the influence of smoking on the overall PFT results. Whether or not systematic differences in smoking habits between the three dust exposure groups may have explained some of the observed lung function disparities is impossible to assess with the available data.

5.4.4 Outcome

Interpretation of pulmonary function test results is highly dependent on whether or not the test is valid: it has been performed in an accepted and standardized way and sources of potential variability have been controlled as much as possible. There are numerous sources of technical and biological variability in pulmonary function testing (ATS, 1991; Becklake et al,, 1993). Technical factors include the instrument itself (calibration, maintenance), the influence of the technician (coaching, feedback), learning effects (ie improvement in a second PFT due to increased familiarity with the test equipment and procedures), procedural protocols, altitude, and temperature. Biological factors include body and neck positioning, quality of the inspiratory manoeuvre, recent activities or exposures, circadian effects, seasonal effects, and health status (in the past and at the time of the PFT). If these sources of variation are not adequately addressed, it is very difficult to differentiate the signal (ie the source of variation of interest, such as dust exposure) from the noise (all other sources of variation) in the PFT results (Becklake et al., 1993). Subsequently, the ability of the PFT to detect changes due to dust exposure is diminished.

Pulmonary function tests are highly effort dependent and the interactions between technicians and subjects are crucial to obtain adequate results; technicians must be trained and must maintain a high level of proficiency to assure optimal results (ATS, 1995). Failure to obtain full understanding, cooperation, and effort from a subject during any part of the test results in an underestimation of the true pulmonary function (Townsend et al., 2000).

The main outcome of interest in this study was pulmonary function. Previous evaluations of the Fibrosis Program have focussed only on x-ray data due to "great variability in PFT results on the same individual from year to year", which suggested poor quality control of the pulmonary function tests (Alleyne, 1988). In an earlier analysis, 9.1% of submitted pulmonary function tests were identified as unacceptable for one of several reasons: less than three spirometry tracings, evidence of inadequate performance, poor expiratory effort, or a faulty machine (Alleyne, 1982). This sub optimal pulmonary function test quality was mentioned in the Fibrosis Program Information Manual, and those who administered the tests were encouraged to utilize training courses at the Northern or Southern Alberta Institute's of Technology, which were set up in collaboration with the Division of Workers' Health, Safety and Compensation. Performance criteria for spirometers were also outlined (Fibrosis Program Information Manual, 1981). Half of all subjects with two or more PFTs had their first PFT prior to 1981, the year when spirometers performance criteria and technician qualification standards were promulgated. Because of the initial poor PFT quality and technician qualifications in the earlier years of the Fibrosis Program, the initial PFTs of a large proportion of subjects may have underestimated their true performance. Assuming a more valid measurement for their last PFT and typical age-related declines, the observed effect might have been no change in pulmonary function or even improvement with time. In this scenario, any potential negative dust-related effects would be obscured due to the invalid first PFT measure.

It has also been noted that longitudinal studies of lung function may be less reliable than cross-sectional analyses, given that PFT results derived from two assessments suffer from twice the degree of measurement variation (Attfield et al., 1996b). For this reason, it is crucial that painstakingly stringent PFT quality control requirements are established when more than one measure of pulmonary function per subject is to be obtained, which has been described previously (Enright et al., 1991; Townsend et al., 1986; Wang et al., 2000b). Data on quality control measures that may have been used in the Fibrosis Program are not available. For example, it is not known if the technician was adequately trained, if the spirometer was properly calibrated and maintained, if the technician, testing equipment, and test protocol were the same for the first and last PFT, if the first and last PFT were performed at the same time of day and time of year, if the subjects were in a similar state of health at the first and last PFT, or if numerous other factors that could have affected PFT results apart from true changes due to dust effects were adequately controlled. Without this information, conclusions drawn from comparing PFT results between subjects in the Fibrosis Program are tenuous.

Even with excellent quality control, natural within-subject biological variability must be taken into account when interpreting pulmonary function change over time. Typically, only changes in FEV_1 or FVC that exceed the estimated 15% year-to-year within-subject variability are considered clinically important (ATS, 1991; Townsend, 2005). Because of this variability, it is recommended that PFT measurements should be made over at least a 4 to 6 year period before any "true" changes in pulmonary function can be observed above the natural background noise in the measurement (Townsend, 2005; Vollmer et al., 1993).

In the present study, 35% (n=1465) of subjects with two or more PFTs had less than 5 years separating their first and last PFT, and 10% of subjects (n=409) had less than two years between their first and last PFT. The influence of a shorter duration between PFTs on variability is demonstrated in Figure 11. Annual change in FEV_1 was calculated by subtracting the first FEV_1 value from the last PFT value and dividing that result by the duration, in years, between the first and last PFT. Subjects were split between those with less than 10 years duration between their first and last PFT (n = 2094, restricted to subjects with at least two years duration due to extreme annual change values in those with less than two years duration) and subjects with 10 or more years duration between their first and last PFT (n= 1703). Although the mean annual decline in FEV_1 was 15 mL for both groups, the greater variability in the data for the subjects with shorter duration is evident by the more extreme maximum and minimum annual change values and much larger standard deviation.

Given the high degree of variability inherent in the data for a substantial proportion of subjects, the likely poor PFT quality and technician qualifications in the early years of the Fibrosis Program, and absence of data on PFT quality control, the present PFT results must be interpreted with caution.

5.4.5 Analysis

The present analytical methods were somewhat crude, in that only the first and last PFT were analyzed to compare different dust exposure groups. Among subjects with two or more PFTs, 3077 (73%) had more than two PFTs, and 1656 subjects had 5 or more PFTs. By relying on only two PFT data points (first and last), a substantial quantity of data is ignored. Other studies have used more robust analytical methods to determine lung function change over time, such as simple linear regression for each subject (Carta et al., 1996; Wang et al., 2005). As well, methods for assessing the clinical significance of lung function changes over time using each individual's longitudinal lower limit of normal have also been promulgated (Townsend, 2005). Whether or not a change in analytical methods in the present study would have changed the overall results in unknown. However, the quality of the analytical results is directly related to the quality of the original data. Due to the probable (but indeterminate) poor quality of the Fibrosis Program PFT data, it is doubtful that a more detailed analysis would have offered any greater insight.

5.5 The Alberta Fibrosis Program

As stated previously, the Alberta Fibrosis Program was designed as a surveillance tool; epidemiological study was not necessarily the intent for the collected data. There are very few provincial or state surveillance schemes for pneumoconiosis and the Alberta Fibrosis Program should be recognized for the ambitious project that it was. Ideally, the Program would have collected one of the largest bodies of data on dust-exposed workers. However, as discussed in Section 1.4 and Section 5.4 above, several shortcomings of the Program limited its usefulness as a surveillance tool.

Perhaps the Fibrosis Program could have been improved through stricter enforcement of Program compliance, so as to ensure that enrolment of at-risk workers was as complete as possible and to ensure adequate follow up through subsequent periodic examinations. A more tailored approach to follow-up that acknowledged the known latency of pneumoconiosis could also have been utilized to minimize the examination burden on workers and employers. For example, the baseline examination might have been followed by a periodic examination every five years, shortening to every two years after ten years of workplace exposures.

Earlier dissemination and stricter enforcement of Fibrosis Program protocols and policies would also have benefited the Program. For example, despite the rather sizable number of subjects enrolled in the Program, there was a tremendous amount of missing data, both in terms of specific outcome measures (PFT, x-ray) and supporting information (symptoms, past medical history, previous dust exposure, height, smoking, etc.). Periodic audits of different employers may have identified poorly completed forms, missed evaluation appointments, and so forth, and highlighted the need for improved diligence. A requirement for employers to provide proof of their reliance on certified pulmonary function technicians, plus a requirement for those technicians to regularly demonstrate and document their quality control practices in order to maintain their certification, would likely have fostered more confidence in the validity of the PFT data.

5.6 Summary

Subjects who were exposed to coal dust had slightly poorer pulmonary function, on average, than subjects exposed to silica/other dust and asbestos at the time of their last PFT in the Fibrosis Program. However, differences were modest, and comparison to external referents through the use of percent predicted FEV_1 and FVC values did not indicate any clinically significant impairment in any dust exposure group. The overall crude prevalence of radiographic pneumoconiosis in this cohort was 2.1%, and roughly half of the subjects with radiographic pneumoconiosis were identified at enrolment into the Fibrosis Program. However, there is a strong indication that the prevalence of radiographic pneumoconiosis was overestimated in this study population, although the extent to which this occurred cannot be determined with certainty without a validation study. Overall, the results suggest that subjects who participated in the Alberta Fibrosis Program from 1964 to 1997 and were included in the present analysis did not experience any significant degree of respiratory impairment due to dust exposure.

Nevertheless, the conclusions that can be gleaned from the present analysis are limited for a number of reasons. Noteworthy among these are inadequate follow-up, indeterminate quality control of PFTs, a high degree of PFT variability, inadequate exposure measurement, inability to completely assess dust exposures prior to entering the Fibrosis Program, and unknown influence of confounding variables such as smoking. The issue of potential selection bias is also critical. Within the Fibrosis Program cohort, only 14% of subjects had at least two PFTs, thereby permitting some degree of assessment of the influence of dust exposure on pulmonary function over time. Out of the entire cohort, just under 4% of subjects had at least 10 years duration between their first and last PFTs and adequate data (ie height) to perform multiple linear regression analyses. The representativeness of the present results to all subjects enrolled in the Fibrosis Program is therefore uncertain and no firm conclusions can be made for the entire Fibrosis Program cohort. Additionally, the participation rate of "at risk" Alberta workers in the Fibrosis Program is unknown, although data from other government surveillance programs would suggest that the participation rate was far less than 100%. Therefore, not only is it unclear if the present results are representative of workers in the Fibrosis Program, but it is also unclear if the results are representative of dust-exposed workers in Alberta in general. The overall influence of coal, silica, and asbestos exposure on respiratory impairment and disability in Alberta workers cannot be adequately assessed from available data in the Alberta Fibrosis Program.

7.0 Tables

Table 1: Occupations listed in Alberta Regulations 186/66 and 375/71 requiring inclusion in the Fibrosis Program.

Free Silica Dust and Carbonaceous Materials	Brick and tile manufacture Cement making Concrete making and breaking Demolition Foundry work Glass making Gravel road maintenance Hard rock mining Pottery and ceramics making Quarrying and stone dressing Rock and gravel crushing Sandblasting Steel manufacture Street sweeping Tunnelling Fertilizer manufacturing Coal mining
Asbestos Dust and Asbestiform Materials	Asbestos processors Auto-body workers Construction workers Demolition workers Insulation workers
Organic Dusts	Feed mill operators Grain elevator operators Seed cleaning and processing plant operators Woodworkers

Table 2: Definitions of exposed workers requiring inclusion in the Fibrosis Program.

Regulation	Definition of Exposed Worker
AB Reg 7/82: Asbestos Regulation	A worker who, for at least 30 days in a 12-month period, will likely be exposed to airborne asbestos in an amount equal to or greater than 25% of the 8-hour Occupational Exposure Limit in the Chemical Hazards Regulation
AB Reg 9/82: Silica Regulation	A worker, who, for at least 30 days in a 12-month period, will likely be exposed to airborne silica in excess of 50% of the 8-hour Occupational Exposure Limit set out in the Chemical Hazards Regulation
AB Reg 243/83: Coal Dust Regulation	A worker whose duties require him, for at least 30 days in a 12-month period, to be in that part of a work site where coal mining or coal processing operations are conducted

Table 3: 8-Hour Occupational Exposure Limits for asbestos, silica, and coal dusts.[a]

Regulation	Dust Type	Specific Exposure	8-hour Occupational Exposure Limits	
AB Reg 8/82 (in force in 1982)	Asbestos (fibres greater than 5 μm in length)	Crocidolite	0.2 fibres / cm^3 of air	
		Amosite and tremolite	0.5 fibres / cm^3 of air	
		All other asbestos fibres	2 fibres / cm^3 of air	
			Respirable mass	Total mass
	Silica	Tripoli, quartz, fused silica	0.1 mg / m^3	0.3 mg / m^3
		Cristobalite, silica flour, tridymite	0.05 mg / m^3	0.15 mg / m^3
		Amorphous	2 mg / m^3	5 mg / m^3
			Respirable mass	
	Coal dust	Underground coal mines	5 mg / m^3	
		Surface coal mines and coal processing plants	2 mg / m^3	
			Respirable mass	
AB Reg 242/83 (in force in 1983)	Coal dust (amendment)	All mine operations	2 mg / m^3	
		High risk operations	4 mg / m^3	
AB Reg 393/88 (in force in 1988)	Asbestos (amendment)	Crocidolite	0.2 fibres / cm^3 of air	
		Amosite and tremolite	0.2 fibres / cm^3 of air	
		All other asbestos fibres	0.5 fibres / cm^3 of air	

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[a] As specified in the Chemical Hazards Regulation of the Alberta Occupational Health and Safety Act.

Table 4: Data contained within the original Fibrosis Program data files.

Variable	Subjects Without PFT Information n=21474	Subjects with at least 1 PFT n=7763	Subjects with 2 or more PFTs[b] n=4206
Birth Date	21387 (99.6)[a]	7732 (99.6)	4200 (99.9)
Sex	21414 (99.7)	7740 (99.7)	4200 (99.9)
Province of Birth	1584 (7.4)	3000 (38.6)	1969 (46.8)
Symptoms	60 (0.3)	254 (3.3)	117 (2.8)
Past Illnesses	61 (0.3)	259 (3.3)	130 (3.1)
Occupational History	2369 (11.0)	7762 (100)	4205 (100)
Industry Code	21474 (100)	7763 (100)	4206 (100)
Company Identification Number	21472 (100)	7763 (100)	4206 (100)
Exposure Code	20651 (96.2)	7448 (95.9)	4206 (100)
Height	3 (0.0)	3895 (50.2)	2425 (57.7)
Smoking Information	85 (0.4)	647 (8.3)	321 (7.6)
ILO graded X-ray	8616 (40.1)	5623 (72.4)	3736 (88.8)

[a] Values in the table represent the number of subjects with data entries for a particular variable, expressed in brackets as a percentage of the subject group.

[b] Note that subjects with "2 or more PFTs" are a subset of the "Subjects with at least 1 PFT" group.

Table 5: Industry category at time of enrolment into the Fibrosis Program.

Code	Industry	Subjects Without PFT Data	Subjects with at least 1 PFT	Subjects with 2 or more PFTs [b]
		n=21474	n=7763	n=4206
1	Coal Mining	8732[a]	3736	2054
2	Insulation Company (Asbestos Removal)	2928	1051	583
3	Quarries, Gravel	1419	492	289
4	Construction Company	135	28	11
5	Chemical Manufacturing	274	125	54
6	Government of Alberta or Municipalities, Hospitals	4915	922	602
7	Cement Factories	227	509	221
8	Foundries	285	113	34
9	Fertilizer Plants	281	43	30
10	Demolition	92	14	4
11	Steelworks	747	152	80
12	Power Plant Utility	409	243	159
13	Asphalt Plant	20	5	3
14	Maintenance Company	168	111	29
15	Heavy Machinery Installation, Maintenance, Repair	13	0	0
16	Railway	15	1	0
17	Manufacture Glass Products	118	34	14
18	Manufacture Drilling Mud, Oilfield Maintenance & Construction	5	24	5
19	Publishing and Printing	8	0	0
20	Spray Application of Plastics or Paints	3	29	0
21	Colleges & Education Institutions	261	22	1
98	Other or Missing Data	409	109	33

[a] Values in the table represent the number of subjects in each industry category

[b] Note that subjects with "2 or more PFTs" are a subset of the "Subjects with at least 1 PFT" group.

Table 6: Descriptive comparison of subjects with and without PFT data.

A)

	Subjects with at least 1 PFT	Subjects Without PFT Data
N	7763	21474
Male (%)	7458 (96.4)	20668 (96.5)
Female (%)	282 (3.6)	746 (3.5)
Date of enrolment (s.d.)[a]	1983.60 (7.93)	1979.04 (6.67) **
Age at enrolment (s.d.)	31.60 (9.90)	32.73 (12.01) **
Exposure code present (%)	7448 (95.9)	20651 (96.2)
coal (%)	3768 (50.6)	8679 (42.0) ††
Silica/other dust (%)	2345 (31.5)	7923 (38.4) ††
asbestos (%)	1291 (17.3)	4027 (19.5) ††
MMMF[b] (%)	44 (0.6)	22 (0.1) ††

B)

	Subjects with at least 1 PFT and Height Data	Subjects Without PFT Data or Without Height Data
N	3895	25342
Male (%)	3730 (96.0)	24386 (96.6)
Female (%)	157 (4.0)	871 (3.4)
Date of enrolment (s.d.)[a]	1987.84 (7.12)	1979.09 (6.60) ***
Age at enrolment (s.d.)	32.40 (9.38)	32.44 (11.79)
Exposure code present (%)	3666 (94.1)	24433 (96.4) ††
coal (%)	1833 (50.0)	10614 (43.4) ††
Silica/other dust (%)	1271 (34.7)	8997 (36.8) ††
asbestos (%)	529 (14.4)	4789 (19.6) ††
MMMF[b] (%)	33 (0.9)	33 (0.1) ††

[a] s.d. = standard deviation

[b] MMMF = Man made mineral fibre

†† Chi Square, $p \leq 0.001$
** T-Test, $p \leq 0.001$

Table 7: Descriptive comparisons between dust exposure groups for subjects with at least one PFT.

A) All Subjects	**Coal**	**Silica/Other Dust**	**Asbestos**	**Mean Comparisons**[a]
n	3768	2345	1291	
Male (%)	3606 (96.1)	2288 (97.7)	1262 (97.9)	††
Female (%)	146 (3.9)	54 (2.3)	27 (2.1)	††
Date of enrolment (s.d.)[b]	1982.63 (7.34)	1984.76 (8.39)	1981.40 (6.78)	cs, ca, sa
Age at enrolment (s.d.)	30.60 (9.29)	33.57 (10.42)	30.90 (9.86)	cs, sa
Subjects with > 0 Years Previous Exposure (%)	795 (21.1)	512 (21.8)	371 (28.7)	††
Previous Exposure [c] **(s.d.)**	4.721 (6.234)	8.382 (8.906)	5.396 (7.538)	cs, sa
FEV_1 in L (s.d.)	3.878 (0.813)	3.939 (0.845)	3.931 (0.809)	
FVC in L (s.d.)	4.735 (0.899)	4.848 (0.926)	4.734 (0.916)	cs, sa
FEV_1/FVC% (s.d.)	81.986 (8.793)	81.283 (9.109)	83.347 (9.022)	cs, ca, sa

B) Subjects with Height Data	**Coal**	**Silica/Other Dust**	**Asbestos**	**Mean Comparisons**[a]
n (%)	1833 (48.7)	1271 (54.2)	529 (41.0)	††
Average height (s.d.)	176.56 (7.30)	175.89 (7.51)	175.92 (7.13)	
Subjects with > 0 Years Previous Exposure (%)	446 (24.3)	286 (22.5)	136 (25.7)	
Previous Exposure [c] **(s.d.)**	5.823 (6.854)	8.921 (9.420)	8.959 (8.765)	cs, ca
FEV_1 in L (s.d.)	4.084 (0.790)	4.012 (0.803)	4.186 (0.741)	sa
FVC in L (s.d.)	5.028 (0.915)	4.963 (0.941)	5.025 (0.862)	
FEV_1/FVC% (s.d.)	81.387 (7.393)	81.032 (7.846)	83.555 (7.735)	ca, sa

[a] Unless otherwise indicated, mean values were compared using a one-way ANOVA and Scheffe post-hoc test. Significant differences ($p < 0.017$, Bonferroni adjustment) between group means are identified in the right-most column as follows: **cs**: coal vs. silica/other dust; **ca**: coal vs. asbestos; **sa**: silica/other dust vs. asbestos.

[b] s.d. = standard deviation

[c] Mean duration of previous exposure for those subjects with greater than 0 years previous exposure.

†† Chi Square, $p \leq 0.001$

Table 8: Results of simple linear regression analyses on first PFT parameters.

Independent Variable	n	FEV$_1$ (L)	FVC (L)	FEV$_1$/FVC (%)
Coal[a]	3666	0.018 (-0.033, 0.069)[b]	0.045 (-0.015, 0.104)	-0.412 (-0.907, 0.084)
Silica/other dust [a]	3666	-0.096*** (-0.150, -0.043)	-0.065* (-0.127, -0.003)	-0.858*** (-1.378, -0.338)
Asbestos [a]	3666	0.130*** (0.057, 0.202)	0.023 (-0.061, 0.108)	2.293*** (1.592, 2.994)
Previous Exposure	3895	-0.013*** (-0.018, -0.008)	-0.013*** (-0.019, -0.007)	-0.053* (-0.100, -0.005)
Age	3881	-0.025*** (-0.027, -0.022)	-0.021*** (-0.024, -0.018)	-0.163*** (-0.188, -0.138)
Sex	3887	-0.843*** (-0.965, -0.720)	-1.111*** (-1.252, -0.969)	1.529* (0.320, 2.738)
Height (cm)	3895	0.052*** (0.049, 0.054)	0.069*** (0.065, 0.072)	-0.082*** (-0.114, -0.050)
Date of first PFT	3895	0.018*** (0.014, 0.021)	0.026*** (0.022, 0.030)	-0.068*** (-0.101, -0.035)

[a] Regression coefficients for exposure type indicator variables represent the PFT findings of those subjects with the noted exposure versus subjects from all other exposure groups combined: a positive coefficient value indicates that subjects in that exposure group had a greater PFT value than the other exposure groups combined; a negative coefficient value indicates that subjects in that exposure group had a lesser PFT value than the other exposure groups combined.

[b] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 9: Results of simple linear regression analyses on first PFT parameters for previous dust exposure, within exposure group comparisons.

	FEV$_1$ (L)	FVC (L)	FEV$_1$/FVC (%)
All Subjects (n=3895)	- 0.013*** (-0.018, -0.008)[a]	-0.013*** (-0.019, -0.007)	-0.053* (-0.100, -0.005)
Previous Exposure			
Coal Subjects (n=1833)	-0.009* (-0.018, -0.001)	-0.009 (-0.019, 0.001)	-0.046 (-0.127, 0.034)
Previous Exposure			
Silica/Other Dust Subjects (n=1271)	-0.022*** (-0.029, -0.014)	-0.021*** (-0.030, -0.012)	-0.093* (-0.168, -0.019)
Previous Exposure			
Asbestos Subjects (n=529)	-0.001 (-0.012, 0.009)	-0.003 (-0.015, 0.010)	0.023 (-0.089, 0.135)
Previous Exposure			

[a] Regression coefficient with 95% confidence interval in brackets

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 10: Results of multiple linear regression analyses on first PFT parameters, within exposure group comparisons.

	FEV_1 (L)	FVC (L)	FEV_1/FVC (%)
All Subjects (n=3881)			
Previous Exposure	-0.002 (-0.006, 0.003) [a]	-0.001 (-0.005, 0.004)	-0.019 (-0.067, 0.030)
Age	-0.025 (-0.027, -0.023)***	-0.021 (-0.024, -0.019)***	-0.163 (-0.189, -0.137)***
Sex	-0.541 (-0.650, -0.432)***	-0.663 (-0.784, -0.541)***	0.324 (-0.947, 1.594)
Height (cm)	0.044 (0.041, 0.047)***	0.061 (0.057, 0.064)***	-0.095 (-0.128, -0.061)***
Date of first PFT	0.024 (0.021, 0.027)***	0.031 (0.028, 0.035)***	-0.038 (-0.073, -0.004)*
Coal Subjects (n=1825)			
Previous Exposure	-0.001 (-0.008, 0.007)	-0.003 (-0.011, 0.005)	0.032 (-0.048, 0.113)
Age	-0.023 (-0.026, -0.020)***	-0.017 (-0.021, -0.014)***	-0.187 (-0.226, -0.148)***
Sex	-0.608 (-0.770, -0.447)***	-0.789 (-0.969, -0.609)***	1.393 (-0.439, 3.225)
Height (cm)	0.044 (0.040, 0.048)***	0.059 (0.054, 0.064)***	-0.072 (-0.120, -0.024)**
Date of first PFT	0.033 (0.029, 0.038)***	0.041 (0.036, 0.046)***	-0.004 (-0.055, 0.047)
Silica/Other Dust Subjects (n=1269)			
Previous Exposure	-0.009 (-0.015, -0.002)**	-0.005 (-0.012, 0.003)	-0.088 (-0.165, -0.011)*
Age	-0.026 (-0.030, -0.022)***	-0.025 (-0.029, -0.021)***	-0.119 (-0.164, -0.074)***
Sex	-0.318 (-0.534, -0.103)**	-0.379 (-0.622, -0.135)**	-0.385 (-2.968, 2.199)
Height (cm)	0.045 (0.040, 0.050)***	0.061 (0.055, 0.067)***	-0.092 (-0.151, -0.032)**
Date of first PFT	0.019 (0.014, 0.024)***	0.029 (0.023, 0.035)***	-0.068 (-0.132, -0.004)*
Asbestos Subjects (n=528)			
Previous Exposure	0.004 (-0.005, 0.013)	0.005 (-0.005, 0.015)	-0.006 (-0.120, 0.108)
Age	-0.028 (-0.034, -0.022)***	-0.024 (-0.031, -0.018)***	-0.157 (-0.229, -0.085)***
Sex	-0.905 (-1.363, -0.448)***	-0.807 (-1.323, -0.291)**	-6.049 (-11.704, -0.394)*
Height (cm)	0.044 (0.037, 0.052)***	0.063 (0.054, 0.071)***	-0.144 (-0.235, -0.053)**
Date of first PFT	0.013 (0.005, 0.021)***	0.020 (0.011, 0.029)***	-0.098 (-0.195, -0.002)*

[a] Regression coefficient with 95% confidence interval in brackets

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 11: Results of multiple linear regression analyses on first PFT percent-predicted FEV$_1$ and FVC, within exposure group comparisons.

	FEV$_1$ (% predicted)	**FVC (% predicted)**
All Subjects (n=3881)		
Previous Exposure	0.001 (-0.100, 0.102)[a]	-0.009 (-0.097, 0.079)
Date of first PFT	0.642 (0.571, 0.712)***	0.619 (0.558, 0.681)***
Coal Subjects (n=1825)		
Previous Exposure	0.064 (-0.105, 0.234)	-0.002 (-0.150, 0.146)
Date of first PFT	0.850 (0.743, 0.956)***	0.784 (0.692, 0.877)***
Silica/Other Dust Subjects (n=1269)		
Previous Exposure	-0.190 (-0.349, -0.031)*	0.122 (-0.262, 0.018)
Date of first PFT	0.544 (0.413, 0.675)***	0.560 (0.444, 0.675)***
Asbestos Subjects (n=528)		
Previous Exposure	0.128 (-0.096, 0.352)	0.088 (-0.108, 0.284)
Date of first PFT	0.374 (0.191, 0.557)***	0.397 (0.237, 0.557)***

[a] Regression coefficient with 95% confidence interval in brackets

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 12: Results of multiple linear regression analyses on first PFT parameters, between exposure group comparisons.

	FEV_1 (L)	FVC (L)	FEV_1/FVC (%)
Coal Subjects vs. All Other Subjects			
Coal[a]	-0.017 (-0.059, 0.026)[b]	0.025 (-0.022, 0.072)	-0.799 (-1.291, -0.306)***
Previous Exposure	-0.002 (-0.006, 0.003)	-0.001 (-0.005, 0.004)	-0.022 (-0.072, 0.027)
Age	-0.026 (-0.028, -0.024)***	-0.022 (-0.025, -0.020)***	-0.165 (-0.192, -0.138)***
Sex	-0.511 (-0.635, -0.387)***	-0.627 (-0.765, -0.489)***	0.269 (-1.175, 1.714)
Height (cm)	0.045 (0.042, 0.048)***	0.061 (0.057, 0.064)***	-0.091 (-0.125, -0.056)***
Date of first PFT	0.024 (0.021, 0.027)***	0.032 (0.029, 0.036)***	-0.047 (-0.084, -0.011)*
Silica/Other Dust Subjects vs. All Other Subjects			
Silica/Other Dust[a]	-0.069 (-0.114, -0.025)**	-0.061 (-0.111, -0.011)*	-0.360 (-0.883, 0.162)
Previous Exposure	-0.001 (-0.005, 0.003)	-0.001 (-0.005, 0.004)	-0.014 (-0.064, 0.035)
Age	-0.025 (-0.028, -0.023)***	-0.022 (-0.025, -0.019)***	-0.160 (-0.187, -0.133)***
Sex	-0.522 (-0.646, -0.399)***	-0.628 (-0.766, -0.491)***	0.059 (-1.385, 1.502)
Height (cm)	0.044 (0.041, 0.047)***	0.061 (0.057, 0.064)***	-0.095 (-0.130, -0.060)***
Date of first PFT	0.025 (0.022, 0.028)***	0.033 (0.029, 0.036)***	-0.035 (-0.072, 0.002)
Asbestos Subjects vs. All Other Subjects			
Asbestos[a]	0.152 (0.093, 0.211)***	0.065 (-0.001, 0.131)	2.054 (1.364, 2.744)***
Previous Exposure	-0.002 (-0.006, 0.002)	-0.001 (-0.005, 0.004)	-0.022 (-0.071, 0.028)
Age	-0.026 (-0.028, -0.023)***	-0.022 (-0.025, -0.020)***	-0.158 (-0.185, -0.132)***
Sex	-0.502 (-0.625, -0.379)***	-0.616 (-0.754, -0.478)***	0.274 (-1.163, 1.711)
Height (cm)	0.045 (0.042, 0.048)***	0.061 (0.058, 0.064)***	-0.091 (-0.126, -0.057)***
Date of first PFT	0.025 (0.021, 0.028)***	0.032 (0.029, 0.036)***	-0.032 (-0.068, 0.004)

n = 3655

[a] Regression coefficients for exposure type indicator variables represent the PFT findings of those subjects with the noted exposure versus subjects from all other exposure groups combined: a positive coefficient value indicates that subjects in that exposure group had a greater PFT value than the other exposure groups combined; a negative coefficient value indicates that subjects in that exposure group had a lesser PFT value than the other exposure groups combined.

[b] Regression coefficient with 95% confidence interval in brackets

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 13: Results of multiple linear regression analyses on first PFT percent-predicted FEV_1 and FVC, between exposure group comparisons.

	FEV_1 (% predicted)	**FVC (% predicted)**
Coal Subjects vs. All Other Subjects		
Coal [a]	-0.350 (-1.382, 0.682)[b]	0.551 (-0.348, 1.451)
Previous Exposure	-0.008 (-0.111, 0.095)	-0.014 (-0.103, 0.076)
Date of first PFT	0.642 (0.567, 0.716)***	0.629 (0.565, 0.694)***

Silica/Other Dust Subjects vs. All Other Subjects		
Silica/Other Dust [a]	-1.519 (-2.610, -0.427)**	-1.145 (-2.097, -0.192)*
Previous Exposure	0.006 (-0.097, 0.108)	-0.010 (-0.100, 0.079)
Date of first PFT	0.665 (0.590, 0.740)***	0.639 (0.574, 0.705)***

Asbestos Subjects vs. All Other Subjects		
Asbestos [a]	3.368 (1.914, 4.823)***	1.085 (-0.186, 2.357)
Previous Exposure	-0.012 (-0.114, 0.090)	-0.021 (-0.110, 0.069)
Date of first PFT	0.660 (0.587, 0.734)***	0.629 (0.565, 0.693)***

$n = 3655$

[a] Regression coefficients for exposure type indicator variables represent the PFT findings of those subjects with the noted exposure versus subjects from all other exposure groups combined: a positive coefficient value indicates that subjects in that exposure group had a greater PFT value than the other exposure groups combined; a negative coefficient value indicates that subjects in that exposure group had a lesser PFT value than the other exposure groups combined.

[b] Regression coefficient with 95% confidence interval in brackets

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 14: Comparison of mean first PFT percent-predicted FEV_1 and FVC between exposure groups.

FEV_1 (% Predicted)		
Coal (n=1825)	**Silica/other dust (n=1269)**	**Asbestos (n=528)**
99.58 (16.53)[a]	100.49 (16.74)	102.08 (14.96)

One-way ANOVA for FEV_1: F = 4.929, p = 0.007
Scheffe Post hoc test: mean difference for coal and asbestos, p = 0.009

FVC (% Predicted)		
Coal (n=1825)	**Silica/other dust (n=1269)**	**Asbestos (n=528)**
96.10 (14.50)	96.74 (14.86)	96.23 (13.18)

One-way ANOVA for FVC: F = 0.751, p = 0.472

[a] Mean values with standard deviation in brackets.

Table 15: Descriptive comparisons of subjects with PFT data: subjects with and without two or more PFTs.

A) All Subjects	Subjects with 2 or more PFTs	Subjects with less than 2 PFTs	
n	4206	3557	
Male (%)	4119 (98.1)	3339 (94.3)	††
Female (%)	81 (1.9)	201 (5.7)	††
Date at first PFT (s.d.)[a]	1982.93 (6.53)	1984.40 (9.26)	**
Age at first PFT (s.d.)	31.71 (9.56)	31.51 (10.29)	
Subjects with > 0 years previous exposure (%)	1205 (28.7)	519 (14.6)	††
Previous Exposure[c] (s.d.)	5.841 (6.890)	5.960 (8.962)	
Exposure code present (%)	4206 (100)	3242 (91.1)	††
Coal (%)	2254 (53.6)	1514 (46.7)	††
Silica/other dust (%)	1194 (28.4)	1151 (35.5)	††
Asbestos (%)	736 (17.5)	555 (17.1)	
MMMF[b] (%)	22 (0.5)	22 (0.7)	
First FEV_1 in L (s.d)	3.927 (0.819)	3.902 (0.827)	
First FVC in L (s.d)	4.786 (0.905)	4.777 (0.924)	
FEV_1/FVC% (s.d)	82.140 (8.598)	81.805 (9.176)	

B) Subjects with Height Data	Subjects with 2 or more PFTs	Subjects with less than 2 PFTs	
n (%)	2425 (57.7)	1470 (41.3)	††
Average height (s.d.)	176.112 (7.085)	176.176 (7.855)	
Subjects with > 0 years previous exposure (%)	624 (25.7)	274 (18.6)	††
Previous Exposure[c] (s.d.)	6.876 (7.680)	7.835 (9.295)	
First FEV_1 in L (s.d)	4.077 (0.777)	4.077 (0.800)	
First FVC in L (s.d)	4.969 (0.891)	5.072 (0.947)	**
FEV_1/FVC% (s.d)	82.210 (7.703)	80.571 (7.255)	**
First FEV_1, % predicted (s.d.)	99.346 (16.446)	102.704 (15.883)	**
First FVC, % predicted (s.d.)	95.124 (14.377)	99.176 (13.989)	**

[a] s.d. = standard deviation

[b] MMMF = Man made mineral fibre

[c] Mean duration of previous exposure for those subjects with greater than 0 years previous exposure.

†† Chi Square, $p \leq 0.001$

** T-Test, $p \leq 0.001$

Table 16: Descriptive comparisons between dust exposure groups for subjects with two or more PFTs.

	Coal	Silica/Other Dust	Asbestos	Mean Comparisons[a]
n	2254	1194	736	
Male Subjects (%)	2197 (97.6)	1177 (98.6)	724 (98.6)	
Female Subjects (%)	53 (2.4)	17 (1.4)	10 (1.4)	
Date of first PFT (s.d.)[b]	1983.57 (6.47)	1983.16 (6.83)	1980.36 (5.48)	ca, sa
Date of last PFT (s.d.)	1992.79 (6.06)	1991.56 (6.46)	1989.57 (5.91)	cs, ca, sa
Age at first PFT	30.73 (8.84)	34.07 (10.40)	30.90 (9.56)	cs, sa
Age at last PFT	39.96 (10.21)	42.48 (10.87)	40.13 (10.17)	cs, sa
Years between first and last PFT (s.d.)	9.22 (6.19)	8.41 (5.79)	9.21 (5.56)	cs, sa
Exposure Duration (s.d.)	10.664 (7.406)	10.461 (8.075)	11.035 (8.026)	
Number of PFTs (s.d.)	4.56 (2.40)	4.19 (2.21)	4.33 (2.30)	cs
Subjects with > 0 years previous exposure (%)	653 (29.0)	322 (27.0)	228 (31.0)	
Previous exposure[c] (s.d)	4.972 (6.045)	7.604 (7.882)	5.886 (7.222)	cs, sa
n with height data present (%)	1409 (62.5)	665 (55.7)	336 (45.7)	††
Average height (s.d.)	176.62 (7.05)	175.39 (7.26)	175.28 (6.65)	cs, ca

[a] Unless otherwise indicated, mean values were compared using a one-way ANOVA and Scheffe post-hoc test. Significant differences ($p < 0.017$, Bonferroni adjustment) between group means are identified in the right-most column as follows: **cs**: coal vs. silica/other dust; **ca**: coal vs. asbestos; **sa**: silica/other dust vs. asbestos.

[b] s.d. = standard deviation

[c] Mean duration of previous exposure for those subjects with greater than 0 years previous exposure.

†† Chi Square, $p \leq 0.001$

Table 17: PFT comparisons between dust exposure groups for subjects with two or more PFTs.

A) All Subjects	**Coal**	**Silica/Other Dust**	**Asbestos**	**Mean Comparisons**[a]
n	2254	1194	736	
First PFT				
FEV_1 in L	3.954 (0.807)[b]	3.874 (0.831)	3.917 (0.827)	
FVC in L	4.827 (0.909)	4.760 (0.883)	4.690 (0.919)	ca
FEV_1/FVC%	82.006 (8.119)	81.353 (9.020)	83.789 (9.112)	ca, sa
Last PFT				
FEV_1 in L	3.828 (0.771)	3.741 (0.798)	3.749 (0.786)	cs
FVC in L	4.812 (0.893)	4.729 (0.943)	4.677 (0.919)	ca
FEV_1/FVC%	79.638 (7.862)	79.207 (7.738)	80.174 (7.110)	

A) Subjects with Height Data	**Coal**	**Silica/Other Dust**	**Asbestos**	**Mean Comparisons**[a]
n (%)	1409	665	336	
First PFT				
FEV_1 in L	4.096 (0.776)	3.975 (0.781)	4.176 (0.761)	cs, sa
FVC in L	5.017 (0.902)	4.868 (0.873)	4.956 (0.867)	cs
FEV_1/FVC%	81.825 (7.394)	81.788 (7.905)	84.552 (8.081)	ca, sa
Last PFT				
FEV_1 in L	3.902 (0.722)	3.837 (0.765)	3.940 (0.736)	
FVC in L	4.926 (0.852)	4.861 (0.934)	4.913 (0.869)	
FEV_1/FVC%	79.331 (7.109)	79.120 (6.941)	80.272 (6.346)	

[a] Unless otherwise indicated, mean values were compared using a one-way ANOVA and Scheffe post-hoc test. Significant differences ($p < 0.017$, Bonferroni adjustment) between group means are identified in the right-most column as follows: **cs**: coal vs. silica/other dust; **ca**: coal vs. asbestos; **sa**: silica/other dust vs. asbestos.

[b] Mean values with standard deviation in brackets.

Table 18: Results of simple linear regression analyses on last PFT parameters.

Independent Variable	n	Last FEV_1 (L)	Last FVC (L)	Last FEV_1/FVC (%)
Coal [a]	2425	0.021 (-0.039, 0.081)[b]	0.041 (-0.029, 0.112)	-0.258 (-0.822, 0.306)
Silica/other dust [a]	2425	-0.077* (-0.143, -0.011)	-0.065 (-0.144, 0.013)	-0.440 (-1.063, 0.183)
Asbestos [a]	2425	0.054 (-0.031, 0.139)	0.005 (-0.096, 0.106)	0.966* (0.162, 1.770)
First PFT value	2425	0.662*** (0.635, 0.690)	0.705*** (0.678, 0.733)	0.416*** (0.384, 0.448)
Exposure Duration	2425	-0.018*** (-0.022, -0.015)	-0.018*** (-0.023, -0.014)	-0.078*** (-0.113, -0.043)
Age at last PFT	2421	-0.036*** (-0.039, -0.033)	-0.039*** (-0.042, -0.035)	-0.118*** (-0.147, -0.089)
Sex	2421	-0.840*** (-1.040, -0.640)	-1.090*** (-1.327, -0.852)	0.510 (-1.406, 2.2427)
Height (cm)	2425	0.050*** (0.046, 0.053)	0.069*** (0.065, 0.073)	-0.097*** (-0.136, -0.058)
Date of last PFT	2425	0.012* (0.001, 0.022)	0.013* (0.001, 0.025)	0.014 (-0.082, 0.110)

[a] Regression coefficients for exposure type indicator variables represent the PFT findings of those subjects with the noted exposure versus subjects from all other exposure groups combined: a positive coefficient value indicates that subjects in that exposure group had a greater PFT value than the other exposure groups combined; a negative coefficient value indicates that subjects in that exposure group had a lesser PFT value than the other exposure groups combined.

[b] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 19: Results of simple linear regression analyses on last PFT parameters for exposure duration, within exposure group comparisons.

	Last FEV$_1$ (L)	Last FVC (L)	Last FEV$_1$/FVC (%)
All Subjects (n=2425)			
Exposure Duration	-0.018 *** (-0.022, -0.015)[a]	-0.018*** (-0.023, -0.014)	-0.078*** (-0.113, -0.043)
Coal Subjects (n=1409)			
Exposure Duration	-0.024*** (-0.029, -0.019)	-0.023*** (-0.029, -0.017)	-0.122*** (-0.173, -0.071)
Silica/Other Dust Subjects (n=665)			
Exposure Duration	-0.011** (-0.017, -0.004)	-0.012** (-0.020, -0.004)	-0.017*** (-0.078, 0.044)
Asbestos Subjects (n=336)			
Exposure Duration	-0.019*** (-0.027, -0.010)	-0.019*** (-0.029, -0.009)	-0.070 (-0.144, 0.005)

[a] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 20: Results of multiple linear regression analyses on last PFT parameters, within exposure group comparisons.

	Last FEV_1 (L)	Last FVC (L)	Last FEV_1/FVC (%)
All Subjects (n=2421)			
First PFT value	0.468 (0.439, 0.498)*** [a]	0.488 (0.457, 0.519)***	0.407 (0.375, 0.439)***
Exposure Duration	-0.002 (-0.004, 0.001)	0.003 (0.000, 0.006)*	-0.075 (-0.108, -0.041)***
Age at last PFT	-0.021 (-0.024, -0.019)***	-0.023 (-0.026, -0.021)***	-0.070 (-0.099, -0.040)***
Sex	-0.333 (-0.470, -0.196)***	-0.307 (-0.465, -0.149)***	-1.881 (-3.621, -0.140)*
Height (cm)	0.021 (0.018, 0.024)***	0.0317 (0.028, 0.035)***	-0.093 (-0.129, -0.057)***
Date of last PFT	0.021 (0.015, 0.028)***	0.016 (0.009, 0.024)***	0.157 (0.071, 0.243)***

Coal Subjects (n=1406)			
First PFT value	0.470 (0.431, 0.510)***	0.496 (0.457, 0.535)***	0.409 (0.365, 0.454)***
Exposure Duration	0.002 (-0.002, 0.006)	0.009 (0.004, 0.013)***	-0.097 (-0.150, -0.044)***
Age at last PFT	-0.021 (-0.024, -0.017)***	-0.022 (-0.026, -0.019)***	-0.075 (-0.116, -0.034)***
Sex	-0.342 (-0.507, -0.177)***	-0.300 (-0.485, -0.115)**	-1.522 (-3.680, 0.636)
Height (cm)	0.019 (0.015, 0.024)***	0.031 (0.026, 0.036)***	-0.111 (-0.160, -0.063)***
Date of last PFT	0.001 (-0.012, 0.014)	-0.006 (-0.020, 0.009)	0.070 (-0.095, 0.235)

Silica/Other Dust Subjects (n=665)			
First PFT value	0.498 (0.441, 0.554)***	0.521 (0.456, 0.586)***	0.415 (0.356, 0.474)***
Exposure Duration	-0.012 (-0.005, 0.004)	0.001 (-0.004, 0.007)	-0.008 (-0.064, 0.049)
Age at last PFT	-0.022 (-0.026, -0.017)***	-0.024 (-0.030, -0.019)***	-0.068 (-0.121, -0.016)**
Sex	-0.262 (-0.548, 0.024)	-0.218 (-0.573, 0.137)	-3.359 (-6.906, 0.188)
Height (cm)	0.022 (0.016, 0.028)***	0.031 (0.024, 0.039)***	-0.095 (-0.160, -0.029)**
Date of last PFT	0.029 (0.019, 0.040)***	0.028 (0.015, 0.041)***	0.152 (0.021, 0.282)*

Asbestos Subjects (n=335)			
First PFT value	0.445 (0.368, 0.521)***	0.453 (0.372, 0.533)***	0.364 (0.287, 0.442)***
Exposure Duration	-0.010 (-0.016, -0.004)***	-0.004 (-0.011, 0.002)	-0.129 (-0.200, -0.057)***
Age at last PFT	-0.026 (-0.032, -0.019)***	-0.032 (-0.040, -0.025)***	-0.003 (-0.078, 0.072)
Sex	-0.123 (-0.648, 0.402)	0.068 (-0.528, 0.664)	-4.212 (-10.649, 2.224)
Height (cm)	0.027 (0.019, 0.036)***	0.036 (0.026, 0.046)***	-0.030 (-0.122, 0.063)
Date of last PFT	0.041 (0.027, 0.055)***	0.037 (0.022, 0.053)***	0.197 (0.027, 0.368)*

[a] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 21: Results of multiple linear regression analyses on last PFT percent-predicted FEV_1 and FVC, within exposure group comparisons.

	Last FEV_1 (% predicted)	Last FVC (% predicted)
All Subjects (n=2421)		
First PFT value	0.468 (0.428, 0.507)***[a]	0.513 (0.481, 0.546)***
Exposure Duration	0.110 (0.039, 0.181)**	0.086 (0.027, 0.145)**
Date of last PFT	0.559 (0.370, 0.749)***	0.301 (0.144, 0.458)***
Coal Subjects (n=1406)		
First PFT value	0.503 (0.461, 0.545)***	0.507 (0.466, 0.548)***
Exposure Duration	0.240 (0.144, 0.336)***	0.191 (0.108, 0.274)***
Date of last PFT	0.128 (-0.204, 0.460)	-0.057 (-0.342, 0.229)
Silica/Other Dust Subjects (n=665)		
First PFT value	0.544 (0.485, 0.604)***	0.571 (0.504, 0.638)***
Exposure Duration	0.151 (0.034, 0.267)*	0.062 (-0.049, 0.173)
Date of last PFT	0.780 (0.504, 1.056)***	0.494 (0.230, 0.758)***
Asbestos Subjects (n=335)		
First PFT value	0.501 (0.418, 0.584)***	0.483 (0.395, 0.572)***
Exposure Duration	-0.158 (-0.302, -0.013)*	-0.123 (-0.255, 0.010)
Date of last PFT	1.018 (0.666, 1.369)***	0.549 (0.230, 0.869)***

[a] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 22: Results of multiple linear regression analyses on last PFT parameters, between exposure group comparisons.

	Last FEV_1 (L)	Last FVC (L)	Last FEV_1/FVC (%)
Coal Subjects vs. All Other Subjects			
Coal [a]	-0.057 (-0.097, -0.018)**[b]	-0.082 (-0.128, -0.037)***	0.096 (-0.411, 0.602)
Exposure Duration	-0.002 (-0.004, 0.001)	0.003 (0.000, 0.006)*	-0.075 (-0.109, -0.041)***
First PFT value	0.467 (0.437, 0.496)***	0.488 (0.457, 0.519)***	0.407 (0.375, 0.439)***
Age at last PFT	-0.021 (-0.024, -0.019)***	-0.024 (-0.026, -0.021)***	-0.069 (-0.099, -0.040)***
Sex	-0.324 (-0.460, -0.187)***	-0.293 (-0.451, -0.135)***	-1.897 (-3.640, -0.154)*
Height (cm)	0.022 (0.019, 0.025)***	0.032 (0.029, 0.036)***	-0.093 (-0.129, -0.057)***
Date of last PFT	0.023 (0.017, 0.030)***	0.019 (0.011, 0.027)***	0.154 (0.067, 0.241)***

Silica/Other Dust Subjects vs. All Other Subjects			
Silica/Other Dust [a]	0.041 (-0.002, 0.084)	0.075 (0.025, 0.125)**	-0.278 (-0.829, 0.272)
Exposure Duration	-0.001 (-0.004, 0.001)	0.004 (0.000, 0.007)*	-0.076 (-0.110, -0.042)***
First PFT value	0.469 (0.440, 0.499)***	0.490 (0.459, 0.521)***	0.406 (0.374, 0.439)***
Age at last PFT	-0.021 ([illegible]24, -0.019)***	-0.024 (-0.026, -0.021)***	-0.068 (-0.098, -0.039)***
Sex	-0.329 (-0.465, -0.192)***	-0.300 (-0.458, -0.141)***	-1.906 (-3.647, -0.164)*
Height (cm)	0.021 (0.018, 0.024)***	0.032 (0.028, 0.036)***	-0.094 (-0.130, -0.058)***
Date of last PFT	0.022 (0.015, 0.028)***	0.017 (0.009, 0.025)***	0.155 (0.069, 0.241)***

Asbestos Subjects vs. All Other Subjects			
Asbestos [a]	0.036 (-0.021, 0.093)	0.039 (-0.027, 0.105)	0.074 (-0.657, 0.805)
Exposure Duration	-0.002 (-0.004, 0.001)	0.003 (0.000, 0.006)	-0.075 (-0.109, -0.041)***
First PFT value	0.466 (0.437, 0.496)***	0.487 (0.456, 0.518)***	0.406 (0.374, 0.439)***
Age at last PFT	-0.021 (-0.024, -0.019)***	-0.023 (-0.026, -0.021)***	-0.069 (-0.099, -0.040)***
Sex	-0.331 (-0.468, -0.194)***	-0.305 (-0.463, -0.146)***	-1.875 (-3.617, -0.134)*
Height (cm)	0.021 (0.018, 0.025)***	0.032 (0.028, 0.036)***	-0.093 (-0.128, -0.057)***
Date of last PFT	0.023 (0.016, 0.029)***	0.017 (0.009, 0.025)***	0.159 (0.071, 0.247)***

$n = 2421$

[a] Regression coefficients for exposure type indicator variables represent the PFT findings of those subjects with the noted exposure versus subjects from all other exposure groups combined: a positive coefficient value indicates that subjects in that exposure group had a greater PFT value than the other exposure groups combined; a negative coefficient value indicates that subjects in that exposure group had a lesser PFT value than the other exposure groups combined.

[b] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 23: Results of multiple linear regression analyses on last PFT percent-predicted FEV_1 and FVC, between exposure group comparisons.

	Last FEV_1 (% predicted)	**Last FVC (% predicted)**
Coal Subjects vs. All Other Subjects		
Coal [a]	-1.074 (-2.116, -0.031)*,[b]	-1.163 (-2.090, -0.237)*
Exposure Duration	0.130 (0.065, 0.196)***	0.087 (0.028, 0.146)**
First PFT value	0.506 (0.475, 0.538)***	0.513 (0.481, 0.546)***
Date of last PFT	0.642 (0.463, 0.821)***	0.340 (0.180, 0.500)***

Silica/Other Dust Subjects vs. All Other Subjects		
Silica/Other Dust [a]	0.509 (-0.625, 1.643)	0.799 (-0.210, 1.808)
Exposure Duration	0.132 (0.066, 0.198)***	0.089 (0.031, 0.148)**
First PFT value	0.508 (0.476, 0.540)***	0.514 (0.482, 0.547)***
Date of last PFT	0.608 (0.432, 0.784)***	0.305 (0.148, 0.462)***

Asbestos Subjects vs. All Other Subjects		
Asbestos [a]	1.086 (-0.426, 2.598)	1.012 (-0.330, 2.354)
Exposure Duration	0.125 (0.059, 0.191)***	0.082 (0.023, 0.141)**
First PFT value	0.506 (0.474, 0.537)***	0.513 (0.480, 0.545)***
Date of last PFT	0.638 (0.456, 0.819)***	0.331 (0.169, 0.493)***

n = 2421

[a] Regression coefficients for exposure type indicator variables represent the PFT findings of those subjects with the noted exposure versus subjects from all other exposure groups combined: a positive coefficient value indicates that subjects in that exposure group had a greater PFT value than the other exposure groups combined; a negative coefficient value indicates that subjects in that exposure group had a lesser PFT value than the other exposure groups combined.

[b] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 24: Comparison of mean last PFT percent-predicted FEV_1 and FVC between exposure groups.

Last FEV_1 (% Predicted)		
Coal (n=1406)	**Silica/other dust (n=665)**	**Asbestos (n=335)**
102.24 (14.83)[a]	102.52 (16.12)	103.37 (15.13)

One-way ANOVA for FEV_1: $F = 1.907$, $p = 0.126$

Last FVC (% Predicted)		
Coal (n=1406)	**Silica/other dust (n=665)**	**Asbestos (n=335)**
98.21 (12.81)	98.73 (14.98)	98.62 (13.12)

One-way ANOVA for FVC: $F = 0.359$, $p = 0.783$

[a] Mean values with standard deviation in brackets.

Table 25: Descriptive comparisons of subjects with two or more PFTs: subjects with ten or more years exposure duration versus subjects with less than ten years exposure duration.

	< 10 years Exposure Duration	≥ 10 years Exposure Duration	
n	2236	1970	
male (%)	2169 (97.3)	1950 (99.0)	†††
female (%)	61 (2.7)	18 (1.0)	†††
Date of first PFT (s.d.)[a]	1985.359 (7.162)	1980.169 (4.304)	**
Date of last PFT (s.d.)	1989.831 (7.246)	1994.234 (3.705)	**
Age at first PFT (s.d.)	32.185 (9.825)	31.178 (9.214)	**
Age at last PFT (s.d.)	36.658 (10.089)	45.248 (8.861)	**
Years Between First and Last PFT (s.d.)	4.472 (2.415)	14.069 (4.556)	**
Exposure Duration	4.732 (2.494)	17.347 (5.926)	**
Number of PFTs (s.d.)	2.89 (1.00)	6.13 (2.21)	**
Exposure Type			
Coal (%)	1153 (51.6)	1101 (55.6)	††
Silica/other dust (%)	676 (30.2)	518 (26.3)	††
Asbestos (%)	385 (17.2)	351 (17.8)	
MMMF[b] **(%)**	22 (0.9)	0 (0)	†††
Subjects with > 0 Years Previous Exposure (%)	327 (14.6)	878 (44.6)	†††
Previous Exposure[c] **(s.d.)**	1.777 (1.719)	7.354 (7.458)	**
Subjects with Height Data (%)	1139 (50.1)	1286 (65.3)	†††
Height (s.d.)	176.198 (7.196)	176.035 (6.987)	

[a] s.d. = standard deviation

[b] MMMF = Man made mineral fibre

[c] Mean duration of previous exposure for those subjects with greater than 0 years previous exposure.

†† Chi Square, $p \leq 0.01$
††† Chi Square, $p \leq 0.001$
** T-Test, $p \leq 0.001$

Table 26: Descriptive comparisons between dust exposure groups for subjects with two or more PFTs: subjects with ten or more years exposure duration versus subjects with less than ten years exposure duration.

	All Subjects		Subjects with Height Data	
	< 10 years Exposure Duration	≥ 10 years Exposure Duration	< 10 years Exposure Duration	≥ 10 years Exposure Duration
Coal				
n	1153	1101	611	798
First PFT				
FEV_1 (L)	4.042 (0.830)[a]	3.862 (0.773) ***	4.306 (0.763)	3.936 (0.747) ***
FVC (L)	4.934 (0.963)	4.716 (0.834) ***	5.302 (0.913)	4.799 (0.831) ***
FEV_1/FVC (%)	82.090 (7.788)	81.918 (8.455)	81.417 (6.548)	82.137 (7.971)
Last PFT				
FEV_1 (L)	3.972 (0.773)	3.678 (0.741) ***	4.113 (0.717)	3.740 (0.684) ***
FVC (L)	4.915 (0.919)	4.705 (0.853) ***	5.132 (0.875)	4.769 (0.800) ***
FEV_1/FVC (%)	81.034 (7.923)	78.175 (7.529) ***	80.369 (6.562)	78.537 (7.408) ***
Silica/other dust				
n	676	518	373	292
First PFT				
FEV_1 (L)	3.871 (0.780)	3.878 (0.895)	3.990 (0.726)	3.957 (0.847)
FVC (L)	4.755 (0.841)	4.766 (0.936)	4.912 (0.821)	4.811 (0.933)
FEV_1/FVC (%)	81.397 (8.487)	81.295 (9.680)	81.298 (7.200)	82.413 (8.695)
Last PFT				
FEV_1 (L)	3.820 (0.746)	3.638 (0.850) ***	3.876 (0.707)	3.787 (0.832)
FVC (L)	4.784 (0.869)	4.657 (1.027) *	4.894 (0.828)	4.820 (1.055)
FEV_1/FVC (%)	79.923 (7.149)	78.273 (8.361) ***	79.295 (6.580)	78.897 (7.381)
Asbestos				
n	385	351	140	196
First PFT				
FEV_1 (L)	3.819 (0.821)	4.024 (0.821) ***	4.224 (0.726)	4.142 (0.784)
FVC (L)	4.595 (0.915)	4.794 (0.913) **	5.036 (0.788)	4.899 (0.916)
FEV_1/FVC (%)	83.389 (9.214)	84.227 (8.991)	84.010 (7.649)	84.940 (8.373)
Last PFT				
FEV_1 (L)	3.775 (0.777)	3.721 (0.797)	4.034 (0.708)	3.872 (0.749) *
FVC (L)	4.658 (0.884)	4.699 (0.956)	4.988 (0.804)	4.859 (0.910)
FEV_1/FVC (%)	81.050 (7.320)	79.212 (6.752) ***	80.959 (6.532)	79.781 (6.179)

[a] Mean values with standard deviation in brackets.

* T-Test, $p \leq 0.05$
** T-Test, $p \leq 0.01$
*** T-Test, $p \leq 0.001$

Table 27: Descriptive percent-predicted PFT value comparisons between dust exposure groups for subjects with two or more PFTs: subjects with ten or more years exposure duration versus subjects with less than ten years exposure duration.

	Subjects with Height Data		
	< 10 years Exposure Duration	≥ 10 years Exposure Duration	
Coal			
n	608	798	
First PFT			
FEV_1 (% predicted)	104.553 (15.167)[a]	94.770 (16.166)	***
FVC (% predicted)	100.877 (13.494)	91.054 (13.959)	***
Last PFT			
FEV_1 (% predicted)	103.677 (14.610)	101.138 (14.916)	***
FVC (% predicted)	99.850 (12.969)	96.961 (12.546)	***
Silica/other dust			
n	373	292	
First PFT			
FEV_1 (% predicted)	101.191 (15.436)	96.408 (18.064)	***
FVC (% predicted)	96.800 (13.327)	92.413 (15.325)	***
Last PFT			
FEV_1 (% predicted)	101.854 (15.187)	103.380 (17.247)	
FVC (% predicted)	98.452 (13.116)	99.092 (17.075	
Asbestos			
n	139	196	
First PFT			
FEV_1 (% predicted)	104.116 (15.311)	98.747 (15.809)	**
FVC (% predicted)	97.567 (12.258)	92.653 (14.147)	***
Last PFT			
FEV_1 (% predicted)	103.331 (15.202)	103.394 (15.117)	
FVC (% predicted)	98.946 (12.506)	98.388 (13.560)	

[a] Mean values with standard deviation in brackets.

*	T-Test, $p \leq 0.05$
**	T-Test, $p \leq 0.01$
***	T-Test, $p \leq 0.001$

Table 28: Results of simple linear regression analyses on last PFT parameters for exposure duration, within exposure group comparisons for subjects with ten or more years exposure duration.

	Last FEV$_1$ (L)	**Last FVC (L)**	**Last FEV$_1$/FVC (%)**
All Subjects (n=1286)			
Exposure Duration	-0.014*** (-0.021, -0.008)[a]	-0.018*** (-0.026, -0.011)	-0.003 (-0.068, 0.062)
Coal Subjects (n=798)			
Exposure Duration	-0.006 (-0.016, 0.004)	-0.008 (-0.019, 0.004)	-0.012 (-0.119, 0.095)
Silica/Other Dust Subjects (n=292)			
Exposure Duration	-0.019** (-0.032, -0.006)	-0.027** (-0.043, -0.010)	0.015 (-0.105, 0.134)
Asbestos Subjects (n=196)			
Exposure Duration	-0.022*** (-0.035, -0.010)	-0.027*** (-0.042, -0.012)	-0.027 (-0.133, 0.079)

[a] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 29: Results of multiple linear regression analyses on last PFT parameters, within exposure group comparisons for subjects with ten or more years exposure duration.

	Last FEV_1 (L)	Last FVC (L)	Last FEV_1/FVC (%)
All Subjects (n=1286)			
First PFT value	0.379 (0.338, 0.420)***[a]	0.422 (0.378, 0.466)***	0.314 (0.269, 0.358)***
Exposure Duration	0.003 (-0.002, 0.007)	0.003 (-0.003, 0.008)	0.011 (-0.051, 0.073)
Age at last PFT	-0.029 (-0.033, -0.026)***	-0.033 (-0.037, -0.028)***	-0.095 (-0.141, -0.048)***
Sex	-0.390 (-0.653, -0.126)**	-0.270 (-0.576, 0.037)	-4.527 (-7.997, -1.056)*
Height (cm)	0.026 (0.022, 0.030)***	0.037 (0.031, 0.042)***	-0.100 (-0.153, -0.046)***
Date of last PFT	0.024 (0.011, 0.038)***	0.031 (0.015, 0.046)***	0.023 (-0.151, 0.197)
Coal Subjects (n=798)			
First PFT value	0.403 (0.351, 0.456)***	0.421 (0.368, 0.475)***	0.316 (0.256, 0.377)***
Exposure Duration	0.015 (0.008, 0.022)***	0.017 (0.009, 0.025)***	0.037 (-0.066, 0.139)
Age at last PFT	-0.025 (-0.030, -0.021)***	-0.027 (-0.032, -0.022)***	-0.118 (-0.180, -0.056)***
Sex	-0.362 (-0.684, -0.039)*	-0.295 (-0.660, 0.070)	-2.902 (-7.524, 1.719)
Height (cm)	0.024 (0.018, 0.029)***	0.036 (0.030, 0.043)***	-0.091 (-0.164, -0.018)*
Date of last PFT	0.008 (-0.015, 0.031)	0.013 (-0.012, 0.039)	0.006 (-0.321, 0.333)
Silica/Other Dust Subjects (n=292)			
First PFT value	0.385 (0.296, 0.474)***	0.468 (0.362, 0.574)***	0.311 (0.221, 0.401)***
Exposure Duration	0.002 (-0.007, 0.012)	-0.002 (-0.014, 0.010)	0.008 (-0.033, 0.195)
Age at last PFT	-0.036 (-0.044, -0.027)***	-0.039 (-0.050, -0.028)***	-0.102 (-0.200, -0.005)*
Sex	-0.455 (-1.040, 0.130)	-0.222 (-0.949, 0.505)	-9.335 (-16.332, -2.339)**
Height (cm)	0.024 (0.015, 0.034)***	0.034 (0.021, 0.046)***	-0.163 (-0.269, -0.058)**
Date of last PFT	0.017 (-0.009, 0.043)	0.026 (-0.007, 0.058)	-0.066 (-0.377, 0.244)
Asbestos Subjects (n=196)			
First PFT value	0.319 (0.221, 0.417)***	0.351 (0.251, 0.451)***	0.322 (0.223, 0.420)***
Exposure Duration	-0.012 (-0.020, -0.003)**	-0.009 (-0.018, 0.000)	-0.102 (-0.204, 0.000)
Age at last PFT	-0.038 (-0.047, -0.029)***	-0.048 (-0.058, -0.037)***	0.003 (-0.105, 0.110)
Sex	-0.254 (-0.922, 0.414)	-0.098 (-0.848, 0.653)	-3.351 (-11.388, 4.686)
Height (cm)	0.036 (0.025, 0.047)***	0.045 (0.032, 0.059)***	-0.032 (-0.154, 0.091)
Date of last PFT	0.060 (0.036, 0.083)***	0.069 (0.043, 0.095)***	0.101 (-0.182, 0.383)

[a] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 30: Results of multiple linear regression analyses on last PFT percent-predicted FEV_1 and FVC, within exposure group comparisons for subjects with ten or more years exposure duration.

	Last FEV_1 (% predicted)	**Last FVC (% predicted)**
All Subjects (n=1286)		
First PFT value	0.418 (0.372, 0.464)***[a]	0.447 (0.400, 0.495)***
Exposure Duration	0.048 (-0.077, 0.173)	-0.034 (-0.146, 0.077)
Date of last PFT	0.703 (0.348, 1.058)***	0.502 (0.188, 0.817)**

Coal Subjects (n=798)		
First PFT value	0.429 (0.372, 0.487)***	0.417 (0.360, 0.473)***
Exposure Duration	0.431 (0.238, 0.625)***	0.277 (0.113, 0.441)***
Date of last PFT	0.442 (-0.183, 1.067)	0.298 (-0.230, 0.826)

Silica/Other Dust Subjects (n=292)		
First PFT value	0.424 (0.324, 0.523)***	0.556 (0.444, 0.667)***
Exposure Duration	-0.069 (-0.321, 0.183)	-0.188 (-0.428, 0.051)
Date of last PFT	0.518 (-0.186, 1.223)	0.471 (-0.197, 1.139)

Asbestos Subjects (n=196)		
First PFT value	0.405 (0.291, 0.520)***	0.399 (0.282, 0.517)***
Exposure Duration	-0.393 (-0.612, -0.173)***	-0.326 (-0.527, -0.126)**
Date of last PFT	1.286 (0.682, 1.890)***	0.853 (0.301, 1.404)**

[a] Regression coefficient with 95% confidence interval in brackets.

* $p \le 0.05$
** $p \le 0.01$
*** $p \le 0.001$

Table 31: Results of multiple linear regression analyses on last PFT parameters, between exposure group comparisons for subjects with ten or more years exposure duration.

	Last FEV_1 (L)	**Last FVC (L)**	**Last FEV_1/FVC (%)**
Coal Subjects vs. All Other Subjects			
Coal [a]	-0.069 (-0.128, -0.011)*[b]	-0.084 (-0.152, -0.016)*	-0.175 (-0.947, 0.598)
Exposure Duration	0.002 (-0.003, 0.007)	0.002 (-0.003, 0.008)	-0.009 (-0.053, 0.072)
First PFT value	0.374 (0.333, 0.415)***	0.417 (0.373, 0.461)***	0.313 (0.268, 0.358)***
Age at last PFT	-0.029 (-0.033, -0.026)***	-0.032 (-0.037, -0.028)***	-0.094 (-0.141, -0.047)***
Sex	-0.393 (-0.655, -0.130)**	-0.273 (-0.579, 0.033)	-4.527 (-7.998, -1.056)*
Height (cm)	0.026 (0.022, 0.031)***	0.037 (0.032, 0.043)***	-0.099 (-0.153, -0.045)***
Date of last PFT	0.027 (0.014, 0.041)***	0.034 (0.019, 0.050)***	0.030 (-0.147, 0.207)

Silica/Other Dust Subjects vs. All Other Subjects			
Silica/Other Dust	0.063 (-0.003, 0.129)	0.081 (0.004, 0.158)*	0.172 (-0.701, 1.046)
Exposure Duration	0.002 (-0.003, 0.007)	0.002 (-0.003, 0.008)	0.010 (-0.053, 0.072)
First PFT value	0.378 (0.337, 0.419)***	0.420 (0.376, 0.464)***	0.314 (0.269, 0.359)***
Age at last PFT	-0.029 (-0.033, -0.026)***	-0.033 (-0.037, -0.028)***	-0.095 (-0.141, -0.048)***
Sex	-0.391 (-0.654, -0.128)**	-0.272 (-0.579, 0.034)	-4.530 (-8.001, -1.059)*
Height (cm)	0.026 (0.022, 0.031)***	0.037 (0.032, 0.042)***	-0.099 (-0.153, -0.045)***
Date of last PFT	0.024 (0.011, 0.037)***	0.030 (0.015, 0.046)***	0.022 (-0.152, 0.196)

Asbestos Subjects vs. All Other Subjects			
Asbestos	0.037 (-0.044, 0.117)	0.039 (-0.055, 0.132)	0.076 (-0.989, 1.141)
Exposure Duration	0.002 (-0.002, 0.007)	0.003 (-0.003, 0.008)	0.011 (-0.051, 0.073)
First PFT value	0.377 (0.336, 0.418)***	0.421 (0.377, 0.465)***	0.313 (0.269, 0.358)***
Age at last PFT	-0.029 (-0.033, -0.026)***	-0.032 (-0.037, -0.028)***	-0.094 (-0.141, -0.047)***
Sex	-0.390 (-0.654, -0.127)**	-0.270 (-0.576, 0.037)	-4.525 (-7.997, -1.054)*
Height (cm)	0.026 (0.022, 0.030)***	0.037 (0.032, 0.042)***	-0.100 (-0.153, -0.046)***
Date of last PFT	0.026 (0.012, 0.040)***	0.033 (0.017, 0.048)***	0.026 (-0.155, 0.207)

n = 1286

[a] Regression coefficients for exposure type indicator variables represent the PFT findings of those subjects with the noted exposure versus subjects from all other exposure groups combined: a positive coefficient value indicates that subjects in that exposure group had a greater PFT value than the other exposure groups combined; a negative coefficient value indicates that subjects in that exposure group had a lesser PFT value than the other exposure groups combined.

[b] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 32: Results of multiple linear regression analyses on last PFT percent-predicted FEV_1 and FVC, between exposure group comparisons for subjects with ten or more years exposure duration.

	Last FEV_1 (% predicted)	**Last FVC (% predicted)**
Coal Subjects vs. All Other Subjects		
Coal[a]	-1.806 (-3.409, -0.202)*[b]	-1.767 (-3.184, -0.350)*
Exposure Duration	0.031 (-0.095, 0.157)	-0.051 (-0.163, 0.061)
First PFT value	0.414 (0.368, 0.459)***	0.443 (0.396, 0.490)***
Date of last PFT	0.783 (0.422, 1.145)***	0.581 (0.261, 0.901)***

Silica/Other Dust Subjects vs. All Other Subjects		
Silica/Other Dust	1.320 (-0.495, 3.134)	1.398 (-0.209, 3.005)
Exposure Duration	0.039 (-0.087, 0.165)	-0.044 (-0.156, 0.068)
First PFT value	0.417 (0.371, 0.463)***	0.446 (0.398, 0.493)***
Date of last PFT	0.699 (0.344, 1.054)***	0.497 (0.183, 0.812)**

Asbestos Subjects vs. All Other Subjects		
Asbestos	1.458 (-0.746, 3.662)	1.275 (-0.671, 3.221)
Exposure Duration	0.044 (-0.081, 0.170)	-0.038 (-0.149, 0.074)
First PFT value	0.416 (0.370, 0.462)***	0.446 (0.399, 0.493)***
Date of last PFT	0.773 (0.403, 1.144)***	0.563 (0.235, 0.891)***

n = 1286

[a] Regression coefficients for exposure type indicator variables represent the PFT findings of those subjects with the noted exposure versus subjects from all other exposure groups combined: a positive coefficient value indicates that subjects in that exposure group had a greater PFT value than the other exposure groups combined; a negative coefficient value indicates that subjects in that exposure group had a lesser PFT value than the other exposure groups combined.

[b] Regression coefficient with 95% confidence interval in brackets.

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 33: Comparison of mean last PFT percent-predicted FEV_1 and FVC between exposure groups for subjects with ten or more years exposure duration.

FIRST FEV_1 (% Predicted)		
Coal	**Silica/other dust**	**Asbestos**
94.770 (16.166)[a]	96.408 (18.064)	98.747 (15.809)
One-way ANOVA for FEV_1: F = 4.837, p = 0.008 Scheffe Post hoc test: mean difference for coal and asbestos, p = 0.011		

LAST FEV_1 (% Predicted)		
Coal	**Silica/other dust**	**Asbestos**
101.138 (14.916)	103.380 (17.247)	103.394 (15.117)
One-way ANOVA for FEV_1: F = 3.181, p = 0.042 Scheffe Post hoc test: no significant mean differences		

FIRST FVC (% Predicted)		
Coal	**Silica/other dust**	**Asbestos**
91.054 (13.959)	92.413 (15.325)	92.653 (14.147)
One-way ANOVA for FEV_1: F = 1.584, p = 0.206		

LAST FVC (% Predicted)		
Coal	**Silica/other dust**	**Asbestos**
96.961 (12.546)	99.092 (17.075)	98.388 (13.560)
One-way ANOVA for FVC: F = 2.848, p = 0.058		

n = 798 for coal-exposed subjects
n = 292 for silica/other dust-exposed subjects
n = 196 for asbestos-exposed subjects

[a] Mean values with standard deviation in brackets.

Table 34: Chest radiograph status across dust exposure groups.

		Coal	Silica/ Other Dust	Asbestos	MMMF	No Exposure Code	Total
A	n	12447	10268	5318	66	1138	29237
	No ILO Coded X-rays	7869 (63.2)[a]	4860 (47.3)	2113 (39.7)	0	156 (13.7)	14998 (51.3)
	At Least One ILO Coded X-ray	4578 (36.8)	5408 (52.7)	3205 (60.3)	66 (100)	982 (86.3)	14239 (48.7)
B	n (≥ 1 ILO coded X-ray)	4578	5408	3205	66	982	14239
	Normal	4476 (97.8)	5305 (98.1)	3111 (97.1)	65 (98.5)	975 (99.3)	13932 (97.8)
	Incident Pneumoconiosis[b]	71 (1.6)	52 (1.0)	39 (1.2)	0	0	162 (1.1)
	Pre-existing Pneumoconiosis	31 (0.7)	51 (0.9)	55 (1.7)	1 (1.5)	7 (0.7)	145 (1.0)
C	n (> 1 ILO coded X-ray)	3545	3715	1811	25	103	9199
	Normal	3447 (97.2)	3630 (97.7)	1740 (96.1)	25 (100)	103 (100)	8945 (97.2)
	Incident Pneumoconiosis	71 (2.0)	52 (1.4)	39 (2.2)	0	0	162 (1.8)
	Pre-existing Pneumoconiosis	27 (0.8)	33 (0.9)	32 (1.8)	0	0	92 (1.0)

A) Subject numbers for those with at least one ILO coded x-ray and those without any ILO coded x-rays. **B)** Subject numbers for those with at least one ILO coded x-ray, divided in three categories: Normal, Incident Pneumoconiosis, and Pre-existing Pneumoconiosis. **C)** Subject numbers for those with at least one ILO coded x-ray and who had more than one x-ray, divided into categories as per B).

[a] Values in table are number of subjects, expressed as a percentage of the respective section's column total n.

[b] Note that subject numbers for incident pneumoconiosis can only apply to those subjects with at least two x-rays. Because subjects with only one x-ray are included in part B of the table, the percentage in brackets represents an underestimate of the true proportion of subjects with incident pneumoconiosis. Data for incident pneumoconiosis in part C are more representative of the proportion of "at-risk" subjects with incident pneumoconiosis.

Table 35: Chest radiograph status across PFT groups.

		Subjects Without PFT Data	Subjects with less than 2 PFTs	Subjects with 2 or more PFTs	Total
A	**n**	21474	3557	4206	29239
	No ILO Coded X-rays	12858 (59.9)[a]	1670 (46.9)	470 (11.2)	14998 (51.3)
	At Least One ILO Coded X-ray	8616 (40.1)	1887 (53.1)	3736 (88.8)	14239 (48.7)

B	**n (≥ 1 ILO coded X-ray)**	8616	1887	3736	14239
	Normal	8454 (98.1)	1866 (98.9)	3612 (96.7)	13932 (97.8)
	Incident Pneumoconiosis[b]	57 (0.7)	10 (0.5)	95 (2.5)	162 (1.1)
	Pre-existing Pneumoconiosis	105 (1.2)	11 (0.6)	29 (0.8)	145 (1.0)

C	**n (> 1 ILO coded X-ray)**	4788	683	3728	9199
	Normal	4675 (97.6)	666 (97.5)	3604 (96.7)	8945 (97.2)
	Incident Pneumoconiosis	57 (1.2)	10 (1.5)	95 (2.5)	162 (1.8)
	Pre-existing Pneumoconiosis	56 (1.2)	7 (1.0)	29 (0.8)	92 (1.0)

A) Subject numbers for those with at least one ILO coded x-ray and those without any ILO coded x-rays. **B)** Subject numbers for those with at least one ILO coded x-ray, divided in three categories: Normal, Incident Pneumoconiosis, and Pre-existing Pneumoconiosis. **C)** Subject numbers for those with at least one ILO coded x-ray and who had more than one x-ray, divided into categories as per B).

[a] Values in table are number of subjects, expressed as a percentage of the respective section's column total n.

[b] Note that subject numbers for incident pneumoconiosis can only apply to those subjects with at least two x-rays. Because subjects with only one x-ray are included in part B of the table, the percentage in brackets represents an underestimate of the true proportion of subjects with incident pneumoconiosis. Data for incident pneumoconiosis in part C are more representative of the proportion of "at-risk" subjects with incident pneumoconiosis.

Table 36: Subject frequencies for maximum ILO code and ILO code of the last x-ray in the Fibrosis Program.

	Incident Pneumoconiosis (n = 162)		Pre-existing Pneumoconiosis (n = 145)	
	Maximum ILO Code	**Last ILO Code**	**Maximum ILO Code**	**Last ILO Code**
0/-		47		10
0/0		8		1
0/1				3
1/0 [a]	89	55	52	50
1/1	48	36	54	44
1/2	11	6	18	17
2/1	7	4	6	6
2/2	4	4	6	6
2/3	1		5	5
3/2	2	2	3	2
3/3			1	1
3/+				

[a] ILO codes of 1/0 or greater indicate the presence of radiographic pneumoconiosis

Table 37: Subject frequencies for duration of previous dust exposure prior to identification of pneumoconiosis, divided by exposure type.

		Incident Pneumoconiosis			Pre-existing Pneumoconiosis		
		Coal	Sil/Oth	Asbestos	Coal	Sil/Oth	Asbestos
Duration from Entry into Fibrosis Program (years)[a]	**0.00**	0	0	0	31	51	55
	0.1 – 5.0	11	15	7	0	0	0
	5.1 – 10.0	12	12	9	0	0	0
	10.1 – 20.0	37	19	21	0	0	0
	20.1 +	11	6	2	0	0	0
Previous Exposure History prior to Entry into Fibrosis Program (years)[b]	**0.0**	60	25	22	17	14	21
	10.1 – 20.0	1	1	2	0	0	0
	20.1 +	1	1	2	0	1	0
	No Data	9	25	13	14	36	34
Total Exposure History Before Pneumoconiosis Identified (years)[c]	**0.0**	0	0	0	31	50	55
	0.1 – 5.0	10	15	7	0	0	0
	5.1 – 10.0	12	12	9	0	0	0
	10.1 – 20.0	37	17	17	0	0	0
	20.1 +	12	8	6	0	1	0

[a] Duration from entry into the Fibrosis Program represents the time from the earliest contact with the Fibrosis Program (either the first x-ray, first PFT, or first "Dispdate") until the identification of pneumoconiosis.

[b] Previous exposure history represents the documented exposure history at the time of entry into the Fibrosis Program.

[c] Total exposure history is the sum of exposure in the Fibrosis Program and pre-Program exposure. For the 1 subject with MMMF exposure and 7 subjects without occupational exposure code data (all with pre-existing pneumoconiosis), the total exposure history for each was 0.0 years.

Table 38: Mean first PFT parameters as a function of x-ray status for all subjects with at least one PFT.[a]

	Normal X-rays	Incident Pneumoconiosis	Pre-existing Pneumoconiosis	Absent X-ray Data	Mean Differences[b]
n	5478	105	40	2140	
FEV_1 (L)	4.008 (0.815)	3.378 (0.863)	2.936 (0.816)	3.721 (0.783)	N/I, N/P, N/A, P/A
FVC (L)	4.916 (0.931)	4.283 (0.868)	3.812 (1.019)	4.481 (0.767)	N/I, N/P, N/A, P/A
FEV_1/FVC (%)	81.675 (8.047)	78.879 (12.269)	77.884 (10.585)	83.015 (10.400)	N/A, I/A, P/A

n	3770	56	10	45	
FEV_1 (% predicted)	100.724 (16.251)	91.126 (17.308)	100.069 (16.325)	102.914 (17.345)	N/I, I/A
FVC (% predicted)	96.756 (14.354)	88.406 (14.976)	98.933 (9.274)	97.425 (12.699)	N/I

[a] total n = 7763 for FEV1, FVC and FEV_1/FVC; total n = 3881 for percent-predicted values. Standard deviations are in brackets.

[b] Significant differences ($p < 0.0083$, Bonferroni adjustment) between group means are identified in the right-most column as follows: **N/I**: normal x-rays vs. incident pneumoconiosis; **N/P**: normal x-rays vs. pre-existing pneumoconiosis; **N/A**: normal x-rays vs. absent x-ray data; **I/P**: incident pneumoconiosis vs. pre-existing pneumoconiosis; **I/A**: incident pneumoconiosis vs absent x-ray data; **P/A**: Pre-existing pneumoconiosis vs absent x-ray data.

Table 39: Mean first PFT parameters (A) and last PFT parameters (B) as a function of x-ray status for all subjects with two or more PFTs.[a]

A) First PFT

	Normal X-rays	Incident Pneumoconiosis	Pre-existing Pneumoconiosis	Absent X-ray Data	Mean Differences[b]
n	3612	95	29	470	
FEV$_1$ (L)	3.993 (0.809)[c]	3.414 (0.847)	3.069 (0.800)	3.576 (0.734)	N/I, N/P, N/A
FVC (L)	4.863 (0.907)	4.287 (0.869)	3.934 (0.8980)	4.343 (0.683)	N/I, N/P, N/A
FEV$_1$/FVC (%)	82.217 (8.209)	79.686 (11.530)	78.439 (10.418)	82.275 (10.429)	

n	2357	49	8	7	
FEV$_1$ (% predicted)	99.525 (16.371)	91.3960 (16.581)	102.468 (16.952)	91.175 (26.310)	N/I
FVC (% predicted)	95.254 (14.352)	88.262 (14.733)	97.879 (10.120)	96.375 (14.882)	

B) Last PFT

	Normal X-rays	Incident Pneumoconiosis	Pre-existing Pneumoconiosis	Absent X-ray Data	Mean Differences
n	3612	95	29	470	
FEV$_1$ (L)	3.833 (0.776)	3.155 (0.741)	2.733 (0.768)	3.664 (0.747)	N/I, N/P, N/A, I/A, P/A
FVC (L)	4.836 (0.911)	4.141 (0.871)	3.730 (0.850)	4.414 (0.777)	N/I, N/P, N/A, P/A
FEV$_1$/FVC (%)	79.330 (7.317)	76.127 (7.702)	73.193 (10.482)	83.011 (9.226)	N/I, N/P, N/A, I/A, P/A

n	2357	49	8	7	
FEV$_1$ (% predicted)	102.640 (15.037)	97.441 (20.144)	98.083 (26.384)	103.712 (21.693)	
FVC (% predicted)	98.508 (13.368)	94.260 (16.245)	96.422 (17.858)	101.564 (15.003)	

[a] n = 4206 for FEV$_1$, FVC and FEV$_1$/FVC; n = 2421 for percent-predicted values. Standard deviations are in brackets.

[b] Significant differences ($p < 0.0083$, Bonferroni adjustment) between group means are identified in the right-most column as follows: **N/I**: normal x-rays vs. incident pneumoconiosis; **N/P**: normal x-rays vs. pre-existing pneumoconiosis; **N/A**: normal x-rays vs. absent x-ray data; **I/P**: incident pneumoconiosis vs. pre-existing pneumoconiosis; **I/A**: incident pneumoconiosis vs absent x-ray data; **P/A**: Pre-existing pneumoconiosis vs absent x-ray data.

[c] Mean values with standard deviation in brackets.

Table 40: Mean ages, PFT dates, and duration between the first and last PFT as a function of x-ray status for all subjects with two or more PFTs.

	Normal X-rays	Incident Pneumoconiosis	Pre-existing Pneumoconiosis	Absent X-ray Data	Mean Differences[b]
n	3612	95	29	470	
Age at First PFT (years)	31.51 (9.32)[a]	37.83 (8.67)	43.46 (9.82)	31.32 (10.62)	N/I, N/P, I/A, P/A
Age at Last PFT (years)	41.11 (52.28)	52.28 (8.74)	52.40 (9.99)	34.35 (11.30)	N/I, N/P, N/A, I/A, P/A
Date of First PFT	1983.89 (6.40)	1978.87 (4.40)	1980.19 (5.81)	1976.56 (3.04)	N/I, N/A
Date of Last PFT	1993.48 (4.63)	1993.32 (4.31)	1989.13 (5.75)	1979.59 (2.93)	N/P, N/A, I/P, I/A, P/A
Time Between First and Last PFTs (years)	9.60 (5.87)	14.56 (5.12)	8.94 (5.62)	3.02 (1.80)	N/I, N/A, I/P, I/A, P/A

[a] Mean values with standard deviation in brackets.

[b] Significant differences ($p < 0.0083$, Bonferroni adjustment) between group means are identified in the right-most column as follows: **N/I**: normal x-rays vs. incident pneumoconiosis; **N/P**: normal x-rays vs. pre-existing pneumoconiosis; **N/A**: normal x-rays vs. absent x-ray data; **I/P**: incident pneumoconiosis vs. pre-existing pneumoconiosis; **I/A**: incident pneumoconiosis vs absent x-ray data; **P/A**: Pre-existing pneumoconiosis vs absent x-ray data.

Table 41: Smoking status for subjects with PFT data, all exposure groups combined.

A)

	Subjects with at least 1 PFT	Subjects with 2 or more PFTs
Present Smoker	255 (39.4)[a]	111 (34.6)
Ex-Smoker	176 (27.2)	99 (30.8)
Ambiguous Smoker	27 (4.2)	11 (3.4)
Never Smoker	189 (29.2)	100 (31.2)
Total	647	321

B)

	Subjects with at least 1 PFT	Subjects with 2 or more PFTs
Ever Smoker	458 (70.8)	221 (68.8)
Never Smoker	189 (29.2)	100 (31.2)
Total	647	321

[a] Number of subjects (expressed as a percentage of all subjects with smoking data in brackets).

Table 42: Pulmonary function parameters as a function of smoking status, within exposure group comparisons for subjects with at least one PFT. [a]

	Coal		Silica/Other Dust		Asbestos	
	Ever Smoker	**Never Smoker**	**Ever Smoker**	**Never Smoker**	**Ever Smoker**	**Never Smoker**
n	224	119	134	42	85	22
FEV_1 (L)	3.990[b]	4.044	3.747	3.601	4.088	4.555*
FVC (L)	4.970	5.042	4.855	4.398*	5.041	5.433
FEV_1/FVC(%)	80.366	80.182	77.600	82.668*	81.382	84.274
Subjects with Height Data						
n	212	116	130	42	81	19
FEV_1 (% predicted)	98.928	98.988	97.044	93.063	100.936	107.109
FVC (% predicted)	96.236	96.905	96.537	88.223*	97.484	100.807

[a] T-Tests were used to compare smoking status within each dust exposure group.

[b] Mean values.

* $p < 0.05$

Table 43: Pulmonary function parameters as a function of smoking status, within exposure group comparisons for subjects with at two or more PFTs. [a]

A) First PFT

	Coal		Silica/Other Dust		Asbestos	
	Ever Smoker	Never Smoker	Ever Smoker	Never Smoker	Ever Smoker	Never Smoker
n	140	72	41	16	40	12
FEV_1 (L)	4.043[b]	4.035	3.726	3.788	4.063	4.695*
FVC (L)	4.972	4.986	4.594	4.468	4.894	5.520*
FEV_1/FVC(%)	81.430	80.785	81.188	85.731*	83.293	85.749

Subjects with Height Data						
n	131	69	40	16	37	10
FEV_1 (% predicted)	98.843	96.472	95.930	90.826	99.796	106.050
FVC (% predicted)	95.325	94.629	92.088	84.460	94.559	99.749

B) Last PFT

	Coal		Silica/Other Dust		Asbestos	
	Ever Smoker	Never Smoker	Ever Smoker	Never Smoker	Ever Smoker	Never Smoker
n	140	72	41	16	40	12
FEV_1 (L)	3.662	3.800	3.581	3.587	3.653	4.201*
FVC (L)	4.717	4.848	4.553	4.597	4.733	5.250
FEV_1/FVC(%)	77.586	78.243	78.614	78.371	77.012	80.192

Subjects with Height Data						
n	131	69	40	16	37	10
FEV_1 (% predicted)	98.226	100.435	98.767	94.159	98.419	99.260
FVC (% predicted)	95.333	98.036	95.431	91.658	96.683	96.449

[a] T-Tests were used to compare smoking status within each dust exposure group.
[b] Mean values.

* $p < 0.05$

Table 44: Results of simple linear regression analyses on first PFT parameters for smoking, within exposure group comparisons.

	FEV_1 (L)	FVC (L)	FEV_1/FVC (%)
Coal Subjects (n=329)			
Smoking	0.057 (-0.127, 0.241)	0.068 (-0.137, 0.273)	-0.031 (-1.735, 1.674)
Silica/Other Dust Subjects (n=172)			
Smoking	-0.154 (-0.464, 0.156)	-0.461* (-0.872, -0.050)	4.941*** (2.104, 7.778)
Asbestos Subjects (n=100)			
Smoking	0.439* (0.064, 0.814)	0.347 (-0.121, 0.816)	3.052 (-0.586, 6.689)

[a] "Smoking" was a dichotomous variable: a positive coefficient indicates that never smokers had a higher PFT value than smokers; a negative coefficient indicates that never smokers had a lower PFT value than smokers.

[b] Regression coefficient (95% confidence interval)

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 45: Results of multiple linear regression analyses (that included smoking as a variable) on first PFT parameters, within exposure group comparisons.

	FEV_1 (L)	FVC (L)	FEV_1/FVC (%)
Coal Subjects (n=328)			
Previous Exposure	-0.012 (-0.025, 0.001)[b]	-0.020 (-0.034, -0.007)**	0.073 (-0.064, 0.210)
Age	-0.027 (-0.035, -0.019)***	-0.019 (-0.027, -0.010)***	-0.238 (-0.324, -0.151)***
Sex	-0.741 (-1.303, -0.180)**	-1.052 (-1.659, -0.445)***	3.288 (-2.798, 9.375)
Height (cm)	0.041 (0.030, 0.052)***	0.056 (0.044, 0.067)***	-0.077 (-0.193, 0.039)
Date of first PFT	0.035 (0.022, 0.048)***	0.043 (0.029, 0.056)***	0.021 (-0.117, 0.160)
Smoking	0.005 (-0.149, 0.159)	0.037 (-0.129, 0.204)	-0.663 (-2.333, 1.007)
Silica/Other Dust Subjects (n=172)			
Previous Exposure	-0.003 (-0.016, 0.010)	0.000 (-0.017, 0.017)	-0.032 (-0.180, 0.115)
Age	-0.029 (-0.040, -0.017)***	-0.031 (-0.046, -0.016)***	-0.087 (-0.217, 0.043)
Sex	-0.140 (-0.633, 0.354)	-0.284 (-0.949, 0.382)	1.256 (-4.469, 6.980)
Height (cm)	0.042 (0.027, 0.056)***	0.056 (0.036, 0.075)***	-0.050 (-0.216, 0.117)
Date of first PFT	0.028 (0.010, 0.045)**	0.042 (0.018, 0.066)***	-0.047 (-0.255, 0.161)
Smoking	-0.083 (-0.333, 0.167)	-0.345 (-0.682, -0.007)*	4.665 (1.763, 7.567)**
Asbestos Subjects (n=100)			
Previous Exposure	0.002 (-0.013, 0.017)	-0.001 (-0.020, 0.018)	0.055 (-0.124, 0.234)
Age	-0.025 (-0.039, -0.012)***	-0.019 (-0.036, -0.002)*	-0.188 (-0.347, -0.030)*
Sex	-0.867 (-1.594, -0.140)*	-0.799 (-1.719, 0.122)	-5.212 (-13.883, 3.459)
Height (cm)	0.046 (0.030, 0.062)***	0.065 (0.044, 0.085)***	-0.131 (-0.323, 0.060)
Date of first PFT	0.002 (-0.016, 0.020)	0.009 (-0.014, 0.032)	-0.118 (-0.335, 0.100)
Smoking	0.279 (-0.029, 0.587)	0.179 (-0.211, 0.570)	2.598 (-1.080, 6.276)

[a] "Smoking" was a dichotomous variable: a positive coefficient indicates that never smokers had a higher PFT value than smokers; a negative coefficient indicates that never smokers had a lower PFT value than smokers.

[b] Regression coefficient (95% confidence interval)

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 46: Results of multiple linear regression analyses (with smoking removed from the regression model) on first PFT parameters, within exposure group comparisons.[a]

	FEV_1 (L)	FVC (L)	FEV_1/FVC (%)
Coal Subjects (n=328)			
Previous Exposure	-0.012 (-0.025, 0.001)	-0.021 (-0.034, -0.007)**	0.078 (-0.058, 0.215)
Age	-0.027 (-0.035, -0.019)***	-0.019 (-0.027, -0.010)***	-0.234 (-0.320, -0.148)***
Sex	-0.740 (-1.293, -0.181)**	-1.040 (-1.644, -0.436)***	3.074 (-2.985, 9.133)
Height (cm)	0.041 (0.030, 0.052)***	0.056 (0.044, 0.067)***	-0.078 (-0.194, 0.038)
Date of first PFT	0.035 (0.022, 0.048)***	0.042 (0.029, 0.056)***	0.024 (-0.114, 0.162)
Silica/Other Dust Subjects (n=172)			
Previous Exposure	-0.002 (-0.015, 0.010)	0.002 (-0.015, 0.019)	-0.057 (-0.207, 0.094)
Age	-0.028 (-0.040, -0.017)***	-0.030 (-0.045, -0.015)***	-0.099 (-0.232, 0.034)
Sex	-0.134 (-0.626, 0.359)	-0.257 (-0.929, 0.414)	0.901 (-4.974, 6.775)
Height (cm)	0.042 (0.028, 0.056)***	0.057 (0.037, 0.076)***	-0.065 (-0.236, 0.105)
Date of first PFT	0.028 (0.011, 0.046)**	0.046 (0.021, 0.070)***	-0.093 (-0.305, 0.118)
Asbestos Subjects (n=100)			
Previous Exposure	0.000 (-0.015, 0.015)	-0.002 (-0.021, 0.016)	0.039 (-0.139, 0.218)
Age	-0.027 (-0.040, -0.014)***	-0.020 (-0.037, -0.003)*	-0.205 (-0.363, -0.047)*
Sex	-0.827 (-1.561, -0.093)*	-0.773 (-1.691, 0.145)	-4.841 (-13.540, 3.857)
Height (cm)	0.047 (0.031, 0.063)***	0.065 (0.045, 0.086)***	-0.119 (-0.311, 0.073)
Date of first PFT	0.001 (-0.018, 0.019)	0.008 (-0.015, 0.031)	-0.133 (-0.351, 0.084)

[a] The same subset of subjects with smoking data described in Table 45 were used in these regression analyses, but with smoking removed from the regression model.

[b] Regression coefficient (95% confidence interval)

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 47: Results of simple linear regression analyses on last PFT parameters for smoking, within exposure group comparisons.

	Last FEV_1(L)	**Last FVC (L)**	**Last FEV_1/FVC (%)**
Coal Subjects (n=200)			
Smoking	0.156 (-0.065, 0.376)	0.168 (-0.067, 0.404)	0.418 (-2.081, 2.916)
Silica/Other Dust Subjects (n=56)			
Smoking	0.020 (-0.446, 0.487)	0.046 (-0.511, 0.603)	0.014 (-4.608, 4.635)
Asbestos Subjects (n=47)			
Smoking	0.410 (-0.191, 1.012)	0.375 (-0.323, 1.074)	2.675 (-3.099, 8.450)

[a] "Smoking" was a dichotomous variable: a positive coefficient indicates that never smokers had a higher PFT value than smokers; a negative coefficient indicates that never smokers had a lower PFT value than smokers.

[b] Regression coefficient (95% confidence interval)

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 48: Results of multiple linear regression analyses (that included smoking as a variable) on last PFT parameters, within exposure group comparisons.

	Last FEV_1 (L)	Last FVC (L)	Last FEV_1/FVC (%)
Coal Subjects (n=200)			
First PFT value	0.388 (0.286, 0.489)***	0.502 (0.405, 0.600)***	0.211 (0.062, 0.359)**
Exposure Duration	-0.006 (-0.018, 0.007)	0.003 (-0.010, 0.015)	-0.045 (-0.239, 0.148)
Age at last PFT	-0.027 (-0.036, -0.016)***	-0.022 (-0.031, -0.012)***	-0.232 (-0.383, -0.080)**
Sex	-0.495 (-1.112, 0.122)	-0.588 (-1.195, 0.018)	0.881 (-8.627, 10.389)
Height (cm)	0.022 (0.010, 0.034)***	0.018 (0.005, 0.031)**	0.039 (-0.136, 0.214)
Date of last PFT	0.015 (-0.034, 0.064)	0.043 (-0.005, 0.090)	-0.138 (-0.899, 0.623)
Smoking	0.085 (-0.073, 0.244)	0.108 (-0.047, 0.263)	-0.299 (-2.754, 2.155)

Silica/Other Dust Subjects (n=56)			
First PFT value	0.290 (0.046, 0.535)*	0.213 (-0.027, 0.453)	0.444 (0.177, 0.711)**
Exposure Duration	-0.008 (-0.023, 0.007)	-0.014 (-0.031, 0.004)	0.062 (-0.112, 0.236)
Age at last PFT	-0.033 (-0.055, -0.012)**	-0.038 (-0.063, -0.014)**	-0.156 (-0.383, 0.071)
Sex	-0.505 (-1.229, 0.219)	-0.280 (-1.122, 0.562)	-8.137 (-16.403, 0.129)
Height (cm)	0.012 (-0.012, 0.035)	0.031 (0.002, 0.059)*	-0.219 (-0.468, 0.031)
Date of last PFT	0.009 (-0.034, 0.052)	0.020 (-0.030, 0.070)	-0.177 (-0.674, 0.320)
Smoking	-0.133 (-0.501, 0.235)	-0.057 (-0.489, 0.376)	-3.270 (-7.669, 1.130)

Asbestos Subjects (n=47)			
First PFT value	0.591 (0.309, 0.873)***	0.465 (0.198, 0.731)***	0.854 (0.580, 1.127)***
Exposure Duration	-0.004 (-0.018, 0.009)	-0.007 (-0.022, 0.007)	-0.028 (-0.196, 0.139)
Age at last PFT	-0.019 (-0.036, -0.002)*	-0.027 (-0.045, -0.008)**	0.018 (-0.179, 0.215)
Sex	0.744 (-0.374, 1.862)	0.840 (-0.363, 2.043)	2.712 (-10.442, 15.866)
Height (cm)	0.032 (0.007, 0.057)*	0.047 (0.017, 0.076)**	0.179 (-0.059, 0.416)
Date of last PFT	0.019 (-0.032, 0.070)	0.009 (-0.047, 0.066)	0.469 (-0.145, 1.083)
Smoking	-0.127 (-0.562, 0.308)	-0.242 (-0.715, 0.231)	2.012 (-3.109, 7.134)

[a] "Smoking" was a dichotomous variable: a positive coefficient indicates that never smokers had a higher PFT value than smokers; a negative coefficient indicates that never smokers had a lower PFT value than smokers.

[b] Regression coefficient (95% confidence interval)

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

Table 49: Results of multiple linear regression analyses (with smoking removed from the regression model) on last PFT parameters, within exposure group comparisons.[a]

	Last FEV_1 (L)	Last FVC (L)	Last FEV_1/FVC (%)
Coal Subjects (n=200)			
First PFT value	0.386 (0.284, 0.487)***	0.502 (0.404, 0.599)***	0.212 (0.064, 0.359)**
Exposure Duration	-0.005 (-0.017, 0.008)	0.004 (-0.009, 0.016)	-0.049 (-0.240, 0.142)
Age at last PFT	-0.028 (-0.037, -0.018)***	-0.023 (-0.033, -0.014)***	-0.228 (-0.375, -0.080)**
Sex	-0.470 (-1.085, 0.146)	-0.555 (-1.160, 0.051)	0.788 (-8.666, 10.243)
Height (cm)	0.022 (0.010, 0.034)***	0.018 (0.005, 0.031)**	0.039 (-0.135, 0.213)
Date of last PFT	0.015 (-0.034, 0.064)	0.042 (-0.006, 0.090)	-0.138 (-0.897, 0.621)

Silica/Other Dust Subjects (n=56)			
First PFT value	0.292 (0.049, 0.535)*	0.217 (-0.018, 0.453)	0.386 (0.128, 0.644)**
Exposure Duration	-0.009 (-0.024, 0.006)	-0.014 (-0.031, 0.003)	0.046 (-0.129, 0.220)
Age at last PFT	-0.032 (-0.053, -0.011)**	-0.037 (-0.061, -0.014)**	-0.131 (-0.358, 0.096)
Sex	-0.463 (-1.174, 0.247)	-0.264 (-1.088, 0.561)	-7.219 (-15.490, 1.052)
Height (cm)	0.012 (-0.012, 0.035)	0.030 (0.002, 0.058)*	-0.227 (-0.480, 0.025)
Date of last PFT	0.008 (-0.035, 0.050)	0.020 (-0.030, 0.069)	-0.186 (-0.689, 0.317)

Asbestos Subjects (n=47)			
First PFT value	0.572 (0.300, 0.843)***	0.436 (0.176, 0.697)**	0.862 (0.591, 1.133)***
Exposure Duration	-0.004 (-0.017, 0.010)	-0.007 (-0.021, 0.008)	-0.037 (-0.202, 0.128)
Age at last PFT	-0.019 (-0.035, -0.002)*	-0.025 (-0.042, -0.007)**	0.013 (-0.191, 0.188)
Sex	0.780 (-0.321, 1.881)	0.923 (-0.269, 2.115)	1.824 (-11.064, 14.712)
Height (cm)	0.033 (0.008, 0.058)*	0.048 (0.018, 0.077)**	0.188 (-0.047, 0.423)
Date of last PFT	0.026 (-0.019, 0.070)	0.002 (-0.027, 0.073)	0.365 (-0.185, 0.915)

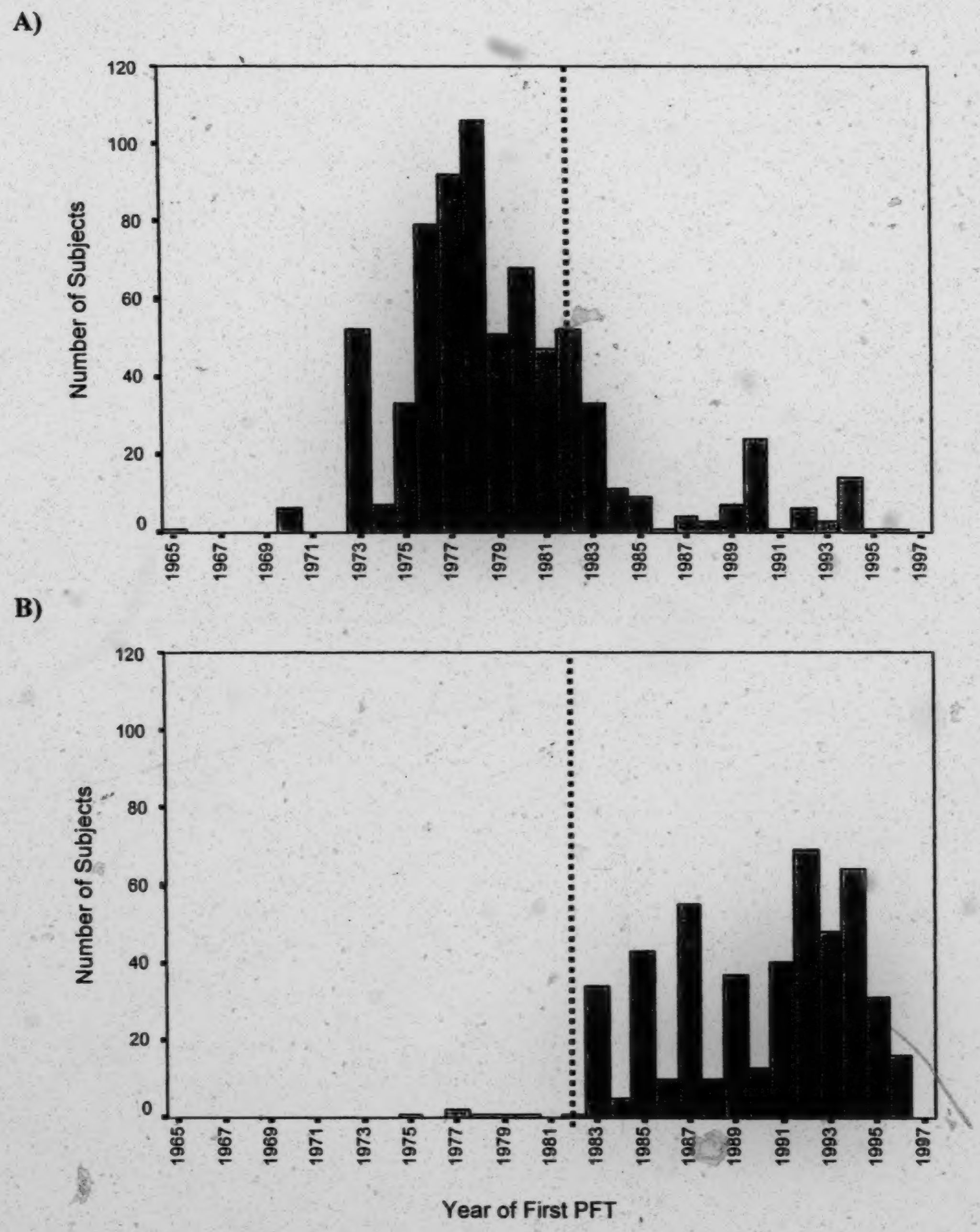
[a] The same subset of subjects with smoking data described in Table 48 were used in these regression analyses, but with smoking removed from the regression model.

[b] Regression coefficient (95% confidence interval)

* $p \leq 0.05$
** $p \leq 0.01$
*** $p \leq 0.001$

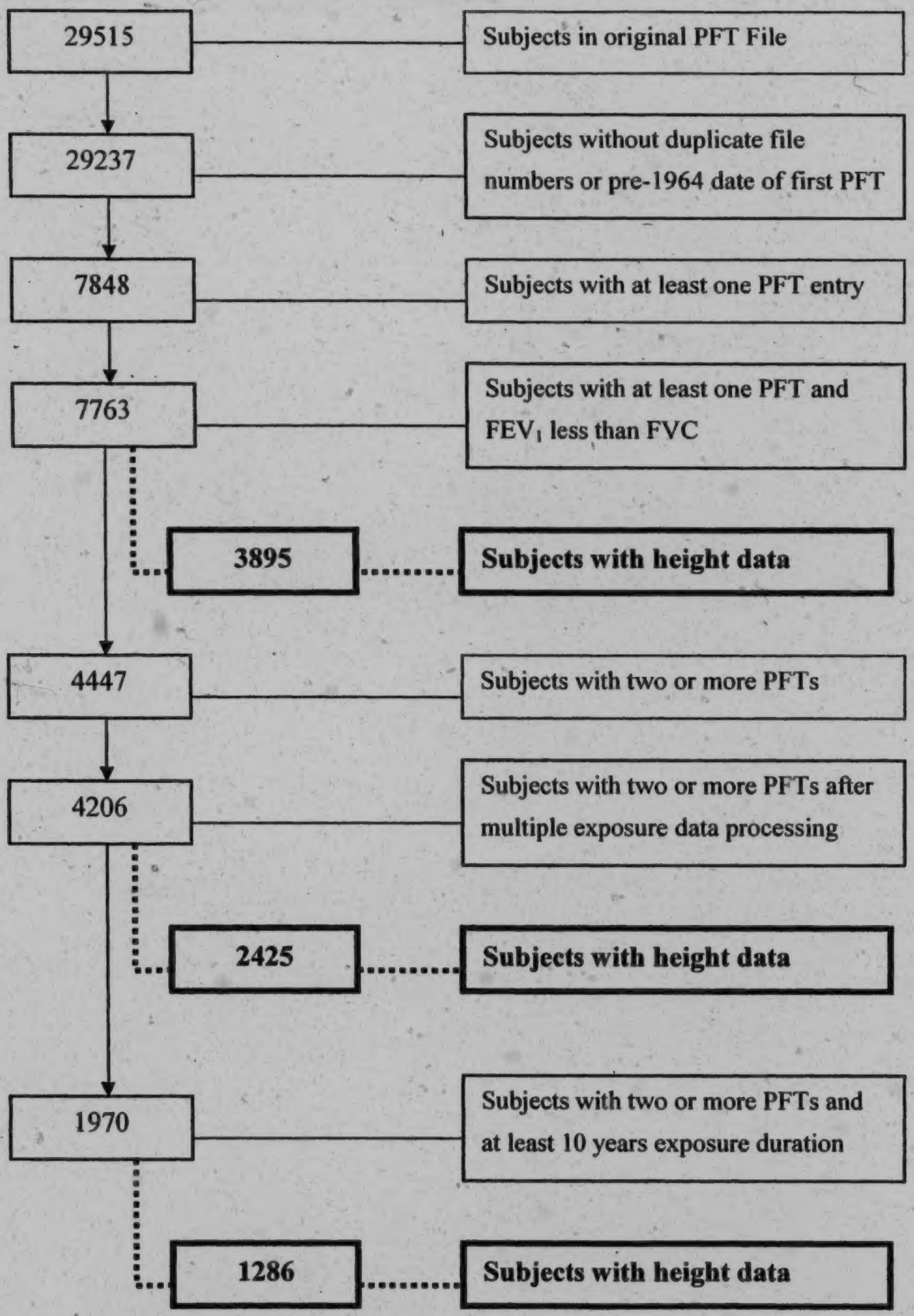
6.0 Figures

Figure 1: Comparison of frequencies of “other dust” and “silica” exposure codes at first PFT.



A) Frequency of subjects whose first PFT was coded as “other dust”. B) Frequency of subjects whose first PFT was coded as “silica”. Note the drop off in the number of subjects coded as “other dust” and corresponding rise in subjects coded as “silica” after 1982 (dashed line).

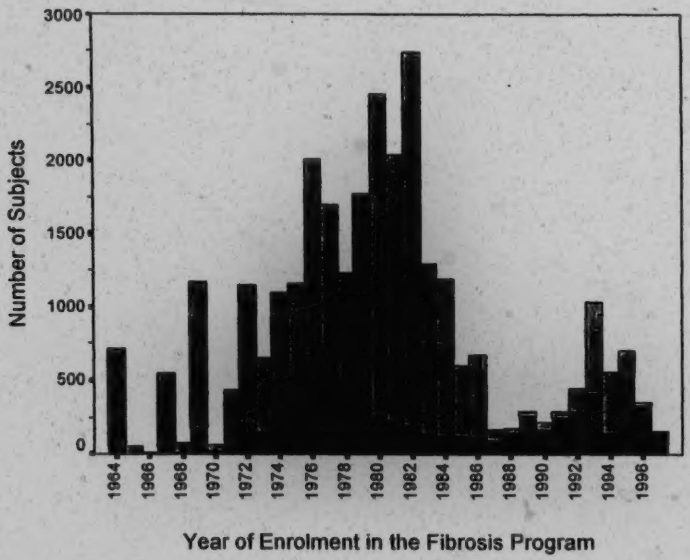
Figure 2: Subject samples available for analysis. [a]


29515
Subjects in original PFT File
29237
Subjects without duplicate file numbers or pre-1964 date of first PFT
7848
Subjects with at least one PFT entry
7763
Subjects with at least one PFT and FEV_1 less than FVC
3895
Subjects with height data
4447
Subjects with two or more PFTs
4206
Subjects with two or more PFTs after multiple exposure data processing
2425
Subjects with height data
1970
Subjects with two or more PFTs and at least 10 years exposure duration
1286
Subjects with height data

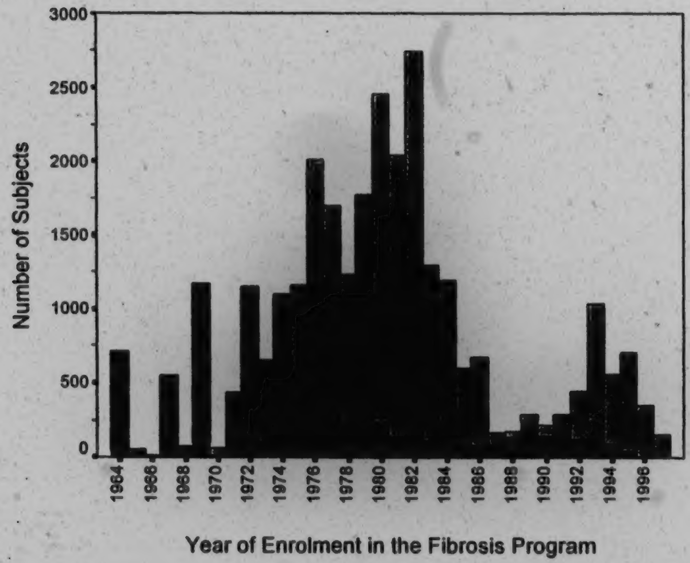
[a] Main analysis subgroups are bolded

Figure 3: Subject enrolment in the Fibrosis Program for all dust exposure types.[a]

A)



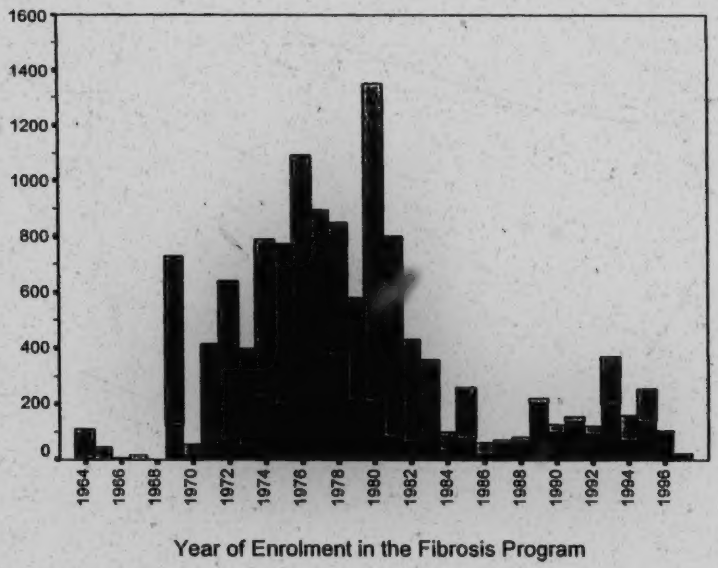
B)



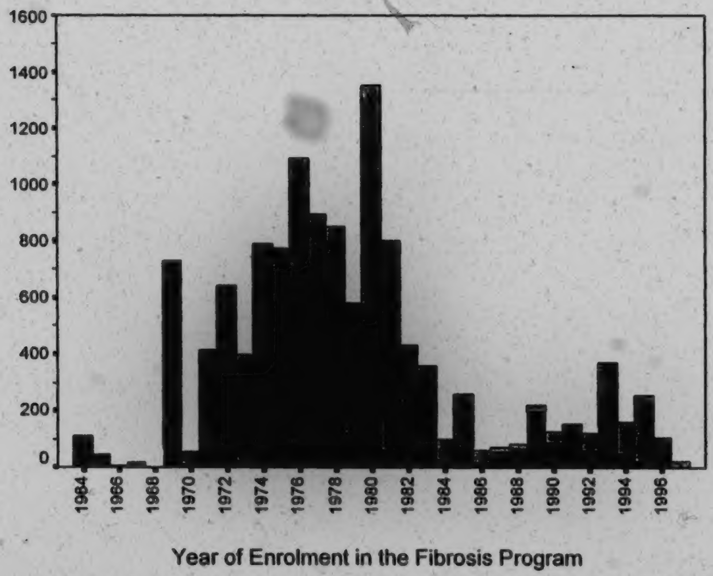
[a] Each stacked bar represents the total number of subjects who entered the Fibrosis Program in a given year (n= 29237 for all subjects). **A)** The black portion of the stacked bars represents the number of subjects with at least one PFT (n=7763) and the grey portion represents the number of subjects who did not have any PFT data and thus were not included in any PFT analyses (n=21470). **B)** The black portion of the stacked bars represents the number of subjects with at least 2 PFTs (n=4206) and the grey portion represents the remaining subjects (n=25031).

Figure 4: Year of enrolment in the Fibrosis Program for subjects with coal exposure.[a]

A)



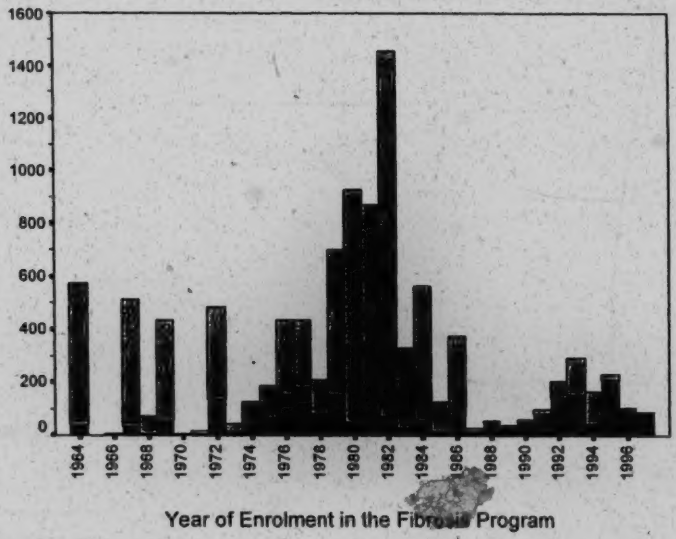
B)



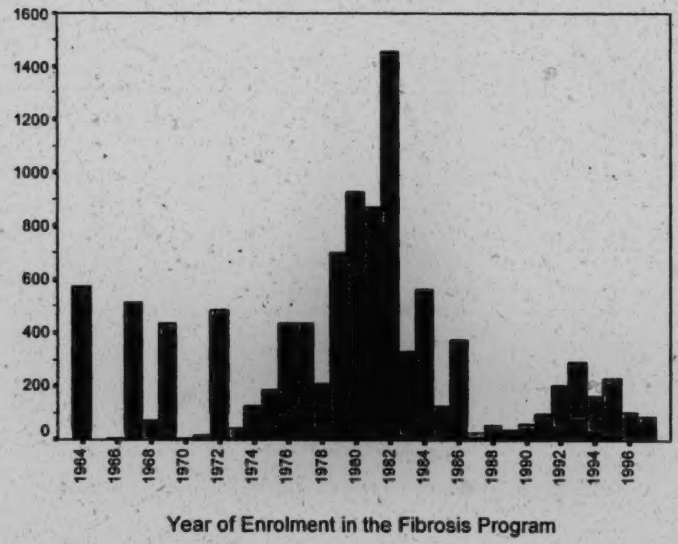
[a] Each stacked bar represents the number of subjects with "Coal" as an exposure code who entered the Fibrosis Program in a given year (n=12447 for all coal-exposed subjects). **A)** The black portion of the stacked bars represents the number of subjects with at least one PFT (n=3768) and the grey portion represents the number of subjects who did not have any PFT data (n=8679). **B)** The black portion of the stacked bars represents the number of subjects with at least 2 PFTs (n=2254) and the grey portion represents the remaining subjects (n=10193).

Figure 5: Year of enrolment in the Fibrosis Program for subjects with silica/other dust exposure.[a]

A)



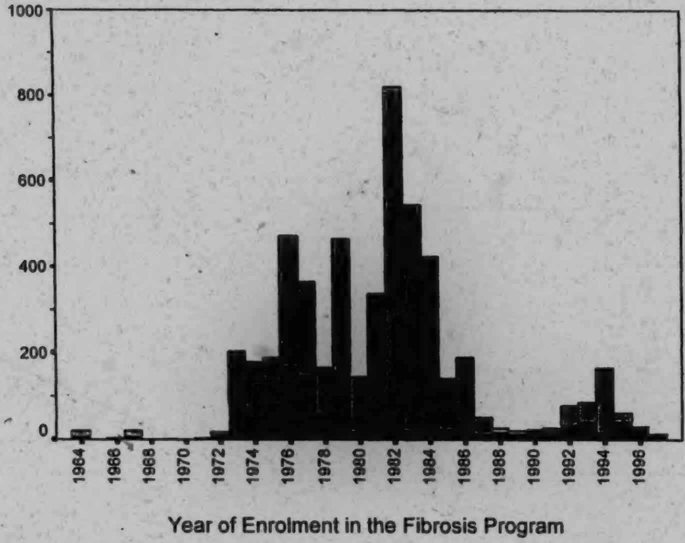
B)



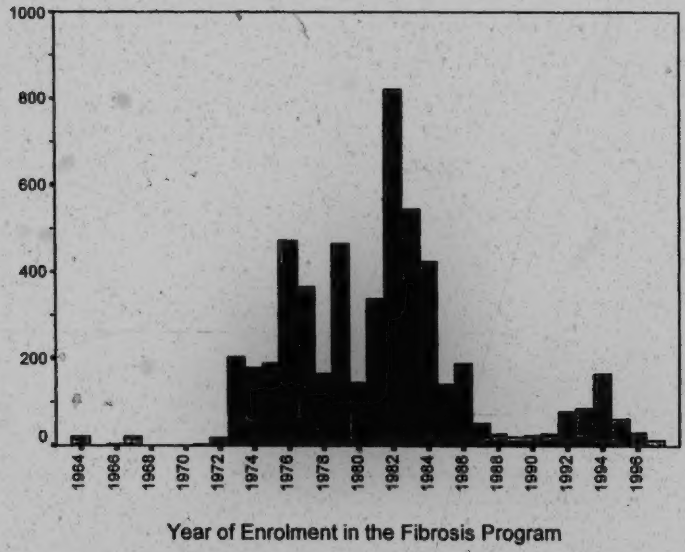
[a] Each stacked bar represents the number of subjects with "Silica/Other Dust" as an exposure code who entered the Fibrosis Program in a given year (n=10268 for all silica/other dust-exposed subjects). **A)** The black portion of the stacked bars represents the number of subjects with at least one PFT (n=2345) and the grey portion represents the number of subjects who did not have any PFT data (n=7923). **B)** The black portion of the stacked bars represents the number of subjects with at least 2 PFTs (n=1194) and the grey portion represents the remaining subjects (n=9074).

Figure 6: Year of enrolment in the Fibrosis Program for subjects with asbestos exposure.[a]

A)

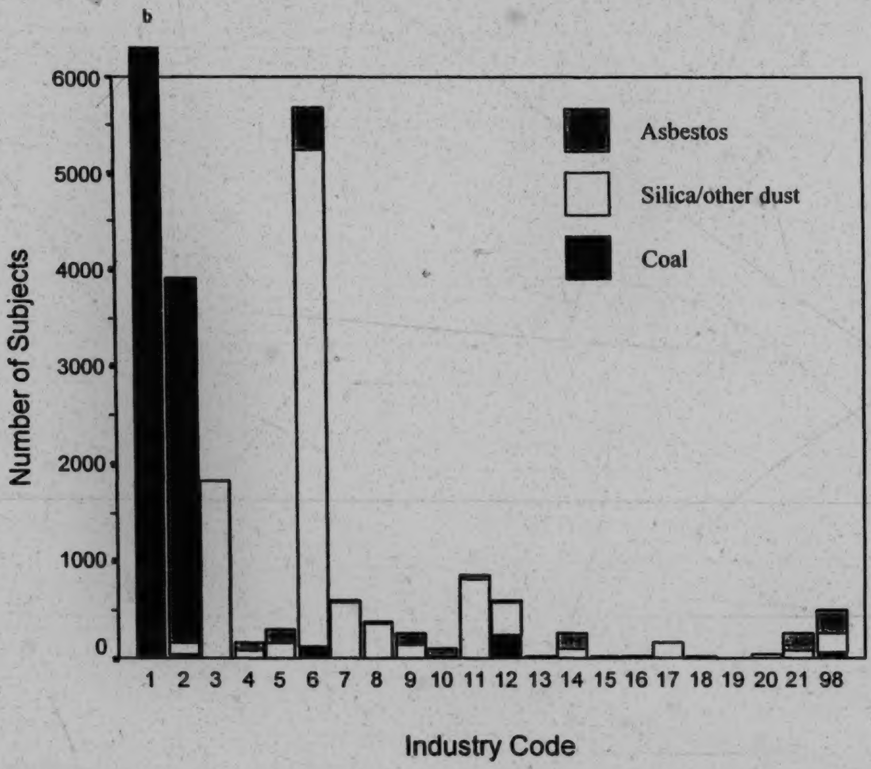


B)



[a] Each stacked bar represents the number of subjects with "Asbestos" as an exposure code who entered the Fibrosis Program in a given year (n=5318 for all asbestos-exposed subjects). **A)** The black portion of the stacked bars represents the number of subjects with at least one PFT (n=1291) and the grey portion represents the number of subjects who did not have any PFT data (n=4027). **B)** The black portion of the stacked bars represents the number of subjects with at least 2 PFTs (n=736) and the grey portion represents the remaining subjects (n=4582).

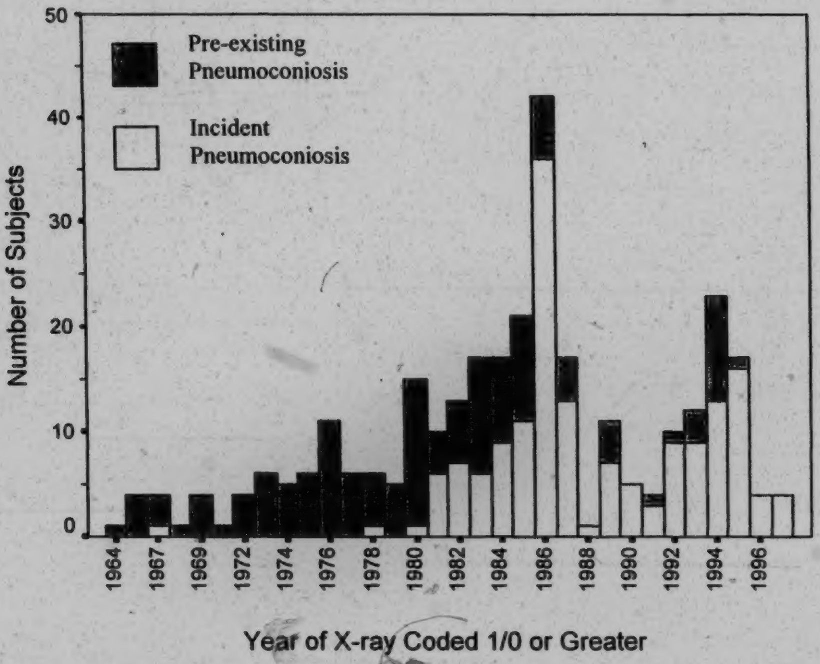
Figure 7: Frequency of subjects with coal, silica/other dust, and asbestos exposure codes for each industry category, all PFT groups combined.[a]



[a] The industry category that corresponds to each numbered industry code on the x-axis is provided in Table 5. Subjects with man made mineral fibre as an exposure or with missing exposure data are not depicted.

[b] Bar truncated. Total number of subjects 12091, of which 11970 coded as "coal", 109 as "silica/other dust", and 12 as "asbestos".

Figure 8: Date of pneumoconiosis identification, all dust types combined.[a]



[a] For subjects with pre-existing pneumoconiosis (gray bars), the year indicates the time of the first x-ray in the Fibrosis Program. For subjects with incident pneumoconiosis (white bars), the year indicates the time of the their first x-ray coded as 1/0 or greater. For the purposes of illustration, all dates are rounded to the nearest year.

Figure 9: Subject age at identification of pneumoconiosis for all subjects with incident pneumoconiosis.

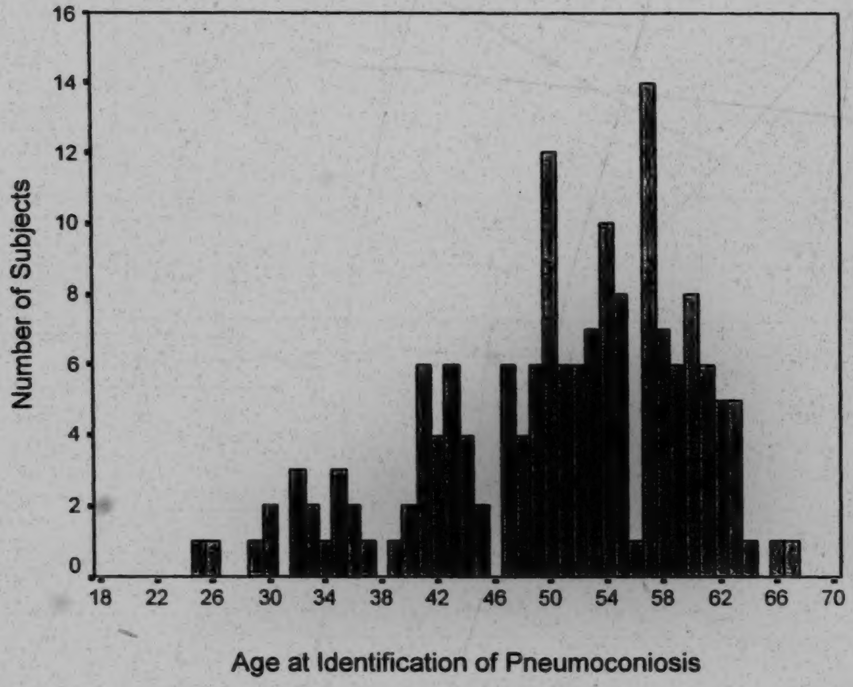


Figure 10: Subject age at identification of pneumoconiosis for all subjects with pre-existing pneumoconiosis.

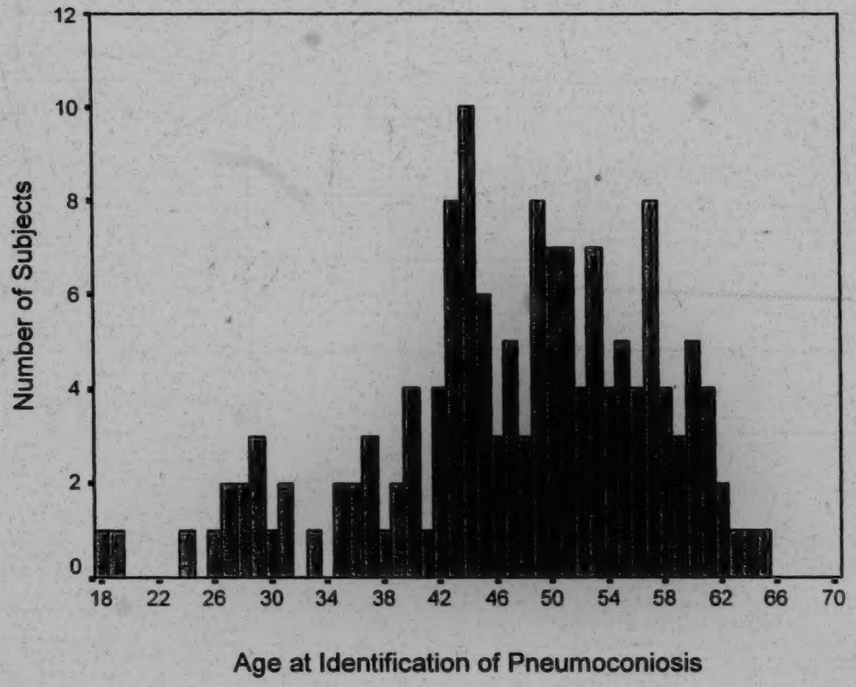
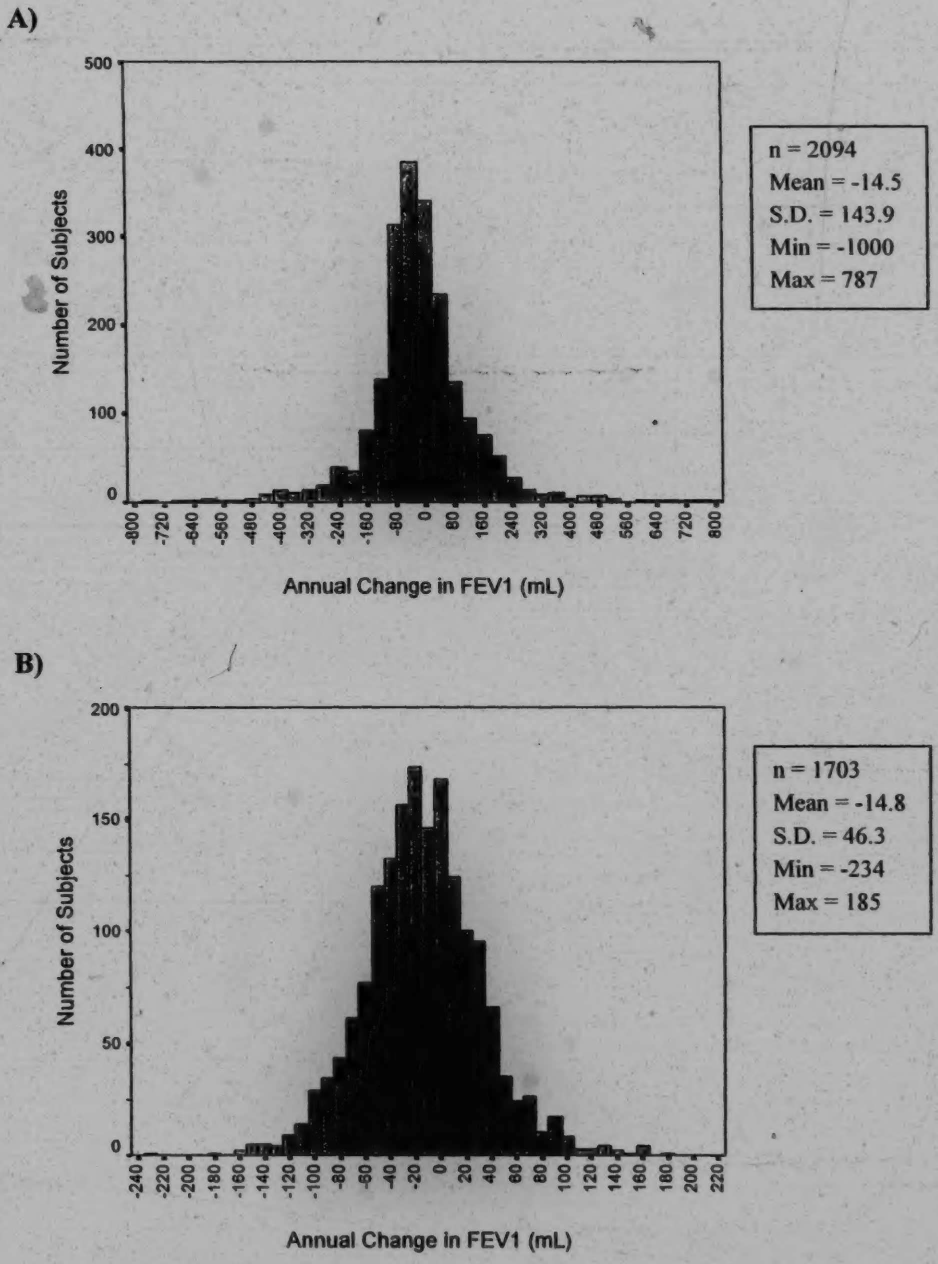


Figure 11: Comparison of PFT variability.



A) Subjects with less than 10 years duration between their first and last PFTs. **B)** Subjects with 10 or more years duration between their first and last PFTs. (Subjects in A restricted to those with at least 2 years between their first and last PFT).

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9.0 Appendices

Appendix 1

International Labour Office Classification of Radiographs of Pneumoconioses

The International Labour Office (ILO) has promulgated a method of classifying postero-anterior chest radiographs for persons with pneumoconiosis (International Labour Office, 1980). The object of the ILO Classification is to codify the radiographic abnormalities of pneumoconiosis in a simple reproducible manner, so as to facilitate international comparisons of pneumoconiosis statistics and research reports. The ILO Classification system has been used extensively in epidemiological research and surveillance of workers in dusty occupations. The most recent guidelines were published in 1980 (International Labour Office, 1980) and are meant to be accompanied by a set of standard radiographs that illustrate the features described. The ILO Classification only addresses radiographic appearance; it does not define pathological entities nor take into account functional capacity.

The ILO Guidelines specify that "there are no features to be seen in a chest radiograph which are pathognomonic of dust exposure". Radiograph readers are instructed to proceed with the ILO Classification of the radiograph if the appearance is consistent with pneumoconiosis, with non-pneumoconiosis findings recorded using symbols and comments. When all of the radiograph abnormalities are due to an etiology other than pneumoconiosis, then the radiograph should is not classified, with the reader opinion only expressed through the appropriate symbols and comments.

After the initial step of recording an impression of the technical quality of the radiograph, the next steps in the ILO Classification are the recognition and recording of both parenchymal and pleural abnormalities, followed by the recording of other radiographic features using symbols and comments.

Parenchymal abnormalities are classified according to location, shape and size, and profusion of opacities. Pleural abnormalities are described by type, location, and approximate dimensions, and extent of calcification.

The most commonly used classification category for the purposes of surveillance and epidemiologic study is profusion. The profusion category is determined by comparing the concentration of opacities in the observed radiograph with the standard radiographs. The opacities are the result of inflammatory changes that occur in the lungs as a result of the pneumoconiosis disease process. The profusion scale is broadly divided into four categories: category 0 indicates that small opacities are absent or less profuse than the lower limit of category 1; categories 1, 2, and 3 represent increasing profusion of small opacities as defined by the corresponding standard radiographs that illustrate the distinctions between categories. In general terms, category 1 indicates that that the small opacities are few in number, category 2 indicates that opacities are numerous but lung marking are still visible, and category 3 indicates that opacities are very numerous and lung markings are obscured.

The 4 broad profusion categories are further subdivided using a 12-point scale consisting of a series of 2-digit combinations. The first digit represents how the radiograph is first classified into one of the four major categories by comparison with the standard radiographs. If during the classification process the major category above or below is seriously considered as an alternative, then this is also recorded as the second digit. For example, category 2/1 represents profusion of major category 2, but with category 1 having been seriously considered as an alternative. Had there been no doubt that the observed radiograph closely matched the category 2 standard radiograph, then the profusion would have been classified as 2/2.

The subdivisions for category 0 are as follows: 0/1 represents profusion of category 0, but category 1 was seriously considered; 0/0 represents a radiograph with either no small opacities, or if opacities are present, they are not sufficiently definite or numerous for category 1 to be considered; 0/- is used when the absence of small opacities is particularly obvious. For category 3, if the observed radiograph shows a markedly higher profusion than would be classified as 3/3, then it is recorded as 3/+.

Thus, the complete 12-point profusion scale is as follows: 0/-, 0/0, 0/1, 1/0, 1/1, 1/2, 2/1, 2/2, 2/3, 3/2, 3/3, 3/+. Category 0/- represents "normal", while the most advanced category is 3/+; the intermediate ten categories are arbitrary divisions of increasing profusion between the two extremes. The concept of a continuum of radiological abnormality for pneumoconiosis is consistent with comparisons between ILO profusion classification and both cumulative dust exposures and the dust content of post-mortem lungs.

Typically, epidemiological studies and surveillance programs have defined radiographic pneumoconiosis as an ILO profusion score of 1/0 or greater or an ILO profusion score of 1/1 or greater.

In the United States, The National Institute for Occupational Safety and Health (NIOSH) certifies physicians in the use of the ILO Classification scheme (no such classification program exists in Canada and most other countries). An "A" reader is an individual who has taken a course given by NIOSH with the assistance of the American College of Radiology. Once that individual successfully passes the certification examination, they are then designated as a "B" reader. This certification lasts for 4 years, after which time a re-certification examination must be completed and passed every 4 years (Henry, 2002).

The ILO classification system was designed for the study of populations for epidemiological and surveillance purposes, with the understanding that some misclassification is acceptable in a population-based study (Welch et al., 1998). However, because of the expected variability in reader interpretation, the Classification can not be used as the sole determinant of disease in an individual case, since qualified readers may differ in the interpretation of a single radiograph (Welch et al., 1998).

Appendix 2

Pulmonary Function Testing

Complete pulmonary function testing consists of the assessment of three main parameters: air flow, lung volumes, and gas transfer. In the majority of studies cited in the present report, the main assessment of interest was that of airflow, typically measured by spirometry. Spirometry measures the volume of air that an individual inhales of exhales as a function of time; flow, or the rate at which the volume is changing as a function of time, may also be measured with spirometry (ATS, 1995). The definitions of the acronyms used to describe pulmonary function parameters that are mentioned in the text of the present report are provided below. Unless otherwise stated, definitions and descriptions were obtained from the following references: ATS, (1995); Miller et al. (2005); Pellegrino et al., 2005; Scanlon et al. (1996). For illustrative purposes, these acronyms are also included in the figures below, reproduced and modified without permission from Townsend et al. (2000).

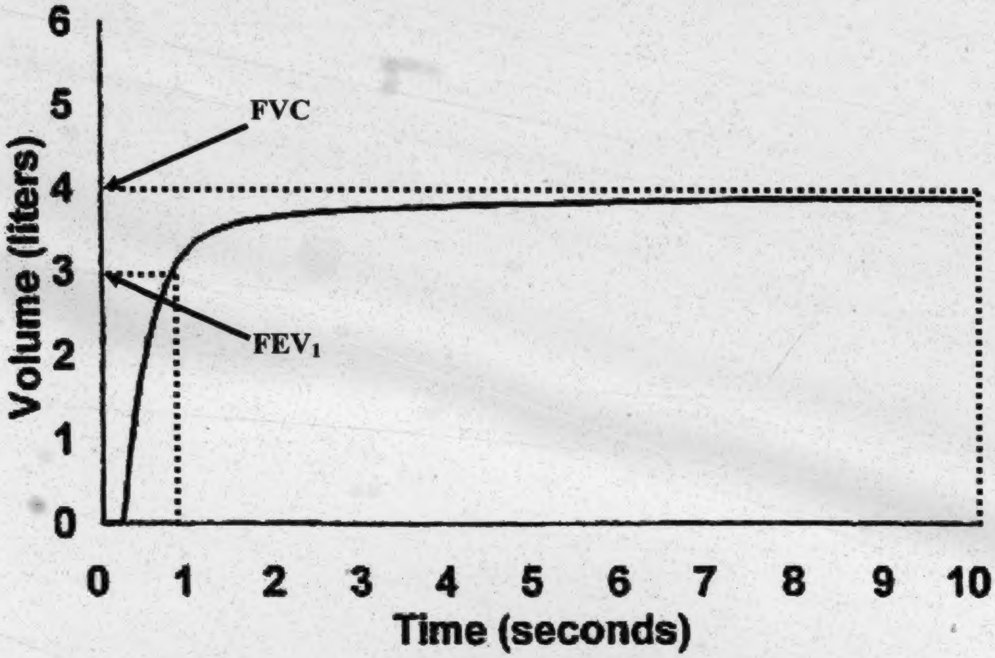
FVC (Forced Vital Capacity) is the maximal volume of air exhaled with maximally forced effort from a position of maximal inspiration, expressed in litres. **VC** (Vital Capacity) is the maximal volume of air exhaled from the point maximal inhalation or the maximal volume of air inhaled from the point of maximal exhalation, and unlike FVC, can be measured with a *slow* exhalation or inhalation. The lungs are never completely "empty" after a maximal exhalation, and the volume of air remaining in the lungs is termed the residual volume (**RV**). The combination of FVC (VC) and RV together make up the **TLC** (Total Lung Capacity).

FEV$_1$ (Forced Expiratory Volume in 1 second) is the volume of air expired in 1 second during the performance of the FVC, expressed in litres.

FEV$_1$/FVC is simply the volume the ratio of the FEV$_1$ volume to the FVC volume, either expressed as a proportion, or when multiplied by 100, as a percent.

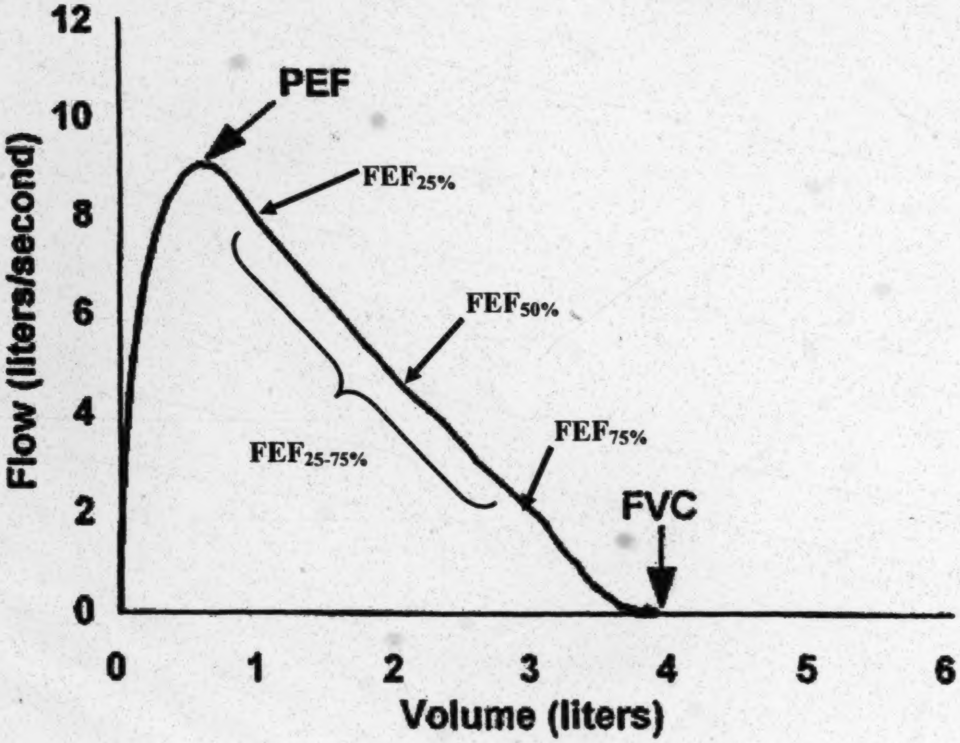
FEF (Forced Expiratory Flow) is a measure of flow rate, expressed in litres/second. Measures of FEF are obtained during the performance of the FVC, and the subscript number indicates the lung volume at which the flow rate was obtained. For example, one of the more common measures for FEF is **FEF$_{25\text{-}75\%}$**, which is the mean forced expiratory flow between 25% and 75% of the FVC. FEF$_{25\text{-}75\%}$ has also been commonly referred to as **MMEF** (Maximum Mid-Expiratory Flow). Other FEF measures include **FEF$_{25\%}$**, **FEF$_{50\%}$**, **FEF$_{75\%}$**, which refer to the maximal flow after 25%, 50%, and 75% of the vital capacity has been expired, respectively. **PEF** (Peak Expiratory Flow) is the maximal flow value during the performance of the FVC.

DLCO (Diffusing Capacity of the Lung for Carbon Monoxide) is a measure of gas transfer that relies on rate of carbon monoxide uptake from the lungs.



Pulmonary function abnormalities observed through spirometry can be broadly divided into obstructive or restrictive. An obstructive ventilatory defect is a disproportionate reduction of maximal airflow from the lung in relation to the maximal volume (ie FVC). It indicates airflow limitation and implies airway narrowing during exhalation. The earliest change associated with flow limitation is thought to be reduction in flow rates at lower than maximal lung volumes, such as in more terminal portions of the spirogram. This will be reflected in measures of instantaneous flow obtained after a portion of FVC has been exhaled, such as $FEF_{25-75\%}$, $FEF_{50\%}$, or $FEF_{75\%}$. As airway disease becomes more advanced and/or more central airways become involved, timed segments of the spirogram, such as the FEV_1, will be reduced out of proportion to the FVC. This will be reflected in a reduction in the FEV_1/FVC.

A restrictive ventilatory defect is characterized physiologically by a reduction in TLC. The presence of a restrictive ventilatory defect may be inferred when the FVC or VC is reduced and the FEV_1/FVC is normal or increased. However, a reduced FVC or VC by itself does not prove that a restrictive ventilatory defect exists, since this can also be caused by submaximal inspiratory or expiratory efforts and/or patchy airflow obstruction.



PEF
FEF25%
FEF50%
FEF75%
FEF25-75%
FVC
Flow (liters/second)
0
2
4
6
8
10
12
Volume (liters)
0
1
2
3
4
5
6

Appendix 3

Regulations and Legislation Pertaining to the Alberta Fibrosis Program

ALBERTA REGULATION 186/66

THE PUBLIC HEALTH ACT

REGULATIONS RESPECTING THE PROTECTION OF PERSONS FROM FIBROSIS OF THE LUNGS

(O. C. 915/66)

(Filed June 1, 1966)

The Lieutenant Governor in Council, upon the recommendation of the Honourable the Minister of Health, pursuant to section 7, subsection (1), clause (*aa*) of The Public Health Act, is pleased to approve Provincial Board of Health Regulations Respecting the Protection of Persons from Fibrosis of the Lungs as follows:

1. Alberta Regulation 572/57 is amended by inserting Division 25 as follows:

2.

DIVISION 25

Regulations Respecting the Protection of Persons from Fibrosis of the Lungs

Application

25-1-1 Every person who is engaged in any occupation where he is or may be exposed to the inhalation of any substance which may produce fibrosis of the lungs shall submit not less than once every two years to an examination to include a 14"x17" chest x-ray and a pulmonary function test.

25-1-2 The foregoing section 25-1-1 shall be considered to apply to any person engaged in any occupation listed hereafter:

-1 Group 1 Occupations: This group shall include those occupations involving exposure of the worker to free silica dusts. These include those involved in:

Street sweeping,
Rock and Gravel Crushing,
Sandblasting,
Hard rock mining,
Coal mining,
Brick, tile, pottery and ceramics making,
Demolition,
Tunnelling,
Cement making,
Foundry work,
Steel making,
Gravel road maintaining,
Concrete making and breaking,
Glass making,
Fertilizer manufacturing.

-2 Group 2 Occupations: This group shall include occupations involving exposure of the worker to asbestos dusts, and shall include the following:

Insulation workers,
Demolition workers,
Asbestos processors.

-3 Group 3 Occupations: This group shall include occupations involving exposure of the worker to organic dusts, and shall include the following:

Feed mill operators,
Seed Cleaning and Processing Plant operators,
Grain Elevator operators.

The Provincial Board of Health may add any occupation to those listed in Group 1, Group 2 or Group 3 above.

25-1-3 Except as provided in section 25-1-4 hereafter every examination required pursuant to this division of the Provincial Board of Health Regulations shall be performed by an official or officials designated by the Provincial Board of Health to conduct such examinations. There shall be no charge to the person examined for the required examination.

25-1-4 Where it is not convenient to have the chest x-ray portion of the examination performed by an official designated pursuant to section 25-1-3, the Provincial Board of Health may arrange for the chest x-ray to be performed in a hospital at the cost of the Department of Public Health. In any such case the hospital superintendent or manager shall cause the x-ray plate to be forwarded to the Director of the Division of Industrial Health Services of the Department of Public Health. In any such case the certificate required under section 25-2-1 shall be issued by the Director of the Division of Industrial Health Services when he is satisfied the entire required examination has been completed.

Certificates and Records

25-2-1 Every person who is examined under the provisions of section 25-1-3 or section 25-1-4 shall be issued a certificate indicating that the examination required by this division of the Provincial Board of Health Regulations has been carried out. The certificate shall show the date of examination and shall be signed by the official designated by the Provincial Board of Health to perform such examination or where applicable by the Director of the Division of Industrial Health Services of the Department of Public Health. Any person receiving the certificate authorized by this section shall furnish the same to the manager, superintendent or other person in charge of the plant or activity employing him.

25-2-2, The manager, superintendent or other person in charge of any plant or activity where any person or persons are engaged in any of the occupations referred to in section 25-1-2 shall obtain from every employee so engaged within two years of his commencing such employment the certificate referred to in section 25-2-1 indicating that the required examination has been carried out. The certificate shall be renewed at intervals which shall be not more than two years apart, except as provided in section 25-2-3 hereafter.

25-2-3 Where due to exigencies which may arise, it may not be practicable for an official designated by the Provincial Board of Health to conduct the required examination, the Provincial Board of Health may direct that the interval between such tests be extended beyond the two year period for such further period as the said Board in its discretion may direct.

25-2-4 Any person who has been issued a certificate under section 25-2-1 indicating that the examination required by this division of the Provincial Board of Health Regulations has been carried out and who has furnished such certificate to the manager, superintendent or other person in charge of the plant or activity and who subsequently severs his employment shall be entitled on request to have such certificate returned to him. If such person is engaged within two years of the date of the certificate in any occupation listed in section 25-1-2 he may furnish such certificate to the manager, superintendent or other person in charge of the plant or activity employing him. In such case the certificate shall continue to be valid for all purposes of this division of the Provincial Board of Health Regulations for a period of two years from the date of the certificate.

25-2-5 The certificate required by section 25-2-2 and furnished under section 25-2-4 shall be kept on file by the manager, superintendent or other person in charge and shall be available for inspection by any Executive Officer of the Local Board of Health or Provincial Board of Health.

25-2-6 The manager, superintendent or other person in charge shall not permit any person to continue in employment contrary to the provisions of sections 25-2-2 and 25-2-4.

Time for Examinations

25-3-1 The manager, superintendent or other person in charge of the plant or activity shall permit any person who is engaged in any occupation listed in section 25-1-2 to undergo the examination required by this division of the Provincial Board of Health Regulations during working hours under such arrangements as may be directed by the official designated by the Provincial Board of Health to conduct such examination. The manager, superintendent or other person in charge shall ensure that any person examined under the provisions of this division receives pay for the time necessary for the examination in the same amount as he would otherwise have received for that time.

Coming Into Force

25-4-1 This division of the Provincial Board of Health Regulations shall come into force July 1, 1966.

REGULATIONS UNDER THE REGULATIONS ACT

ALBERTA REGULATION 187/66

THE TURNER VALLEY UNIT OPERATIONS ACT

OIL AND GAS CONSERVATION BOARD

IN THE MATTER OF TURNER VALLEY UNIT NO. 4

ORDER NO. TVU 41

(Filed June 8, 1966)

Whereas on May 24, 1966, examiners appointed by the Oil and Gas Conservation Board heard an application by Western Decalta Petroleum Limited, as unit operator of Turner Valley Unit No. 4, for amendment of Order No. TVU 4.

Therefore, the Oil and Gas Conservation Board, pursuant to The Turner Valley Unit Operations Act, being chapter 91 of the Statutes of Alberta, 1958, hereby orders as follows:

1. Order No. TVU 4, being Alberta Regulation 210/60, is amended.

2. Schedule V is amended by striking out clause 1 thereof and by substituting the following:

1. (1) Water shall be injected to the Turner Valley Rundle Pool in the wells:

Ang Cdn 7 TV 4 IN 9-31-18-2
Ang Cdn 5 TV 4 IN 8-31-18-2
Command 2 TV 4 IN 11-29-18-2
Royalite 31 TV 4 IN 6-29-18-2
Royalite 36 TV4 IN 13-32-18-2
Turner Valley Royalties #1
B&B Royalites #1
Westflank 1 TV4 IN 1-32-18-2

and in such other wells as the Board may, from time to time, direct or authorize.

(2) Gas shall be injected to the Turner Valley Rundle Pool in the wells:

Globe 1 TV IN 7-29-18-2
Westflank 1 TV4 IN 1-32-18-2
Sterling Pacific No. 5
Westflank Oil Company Limited #3 Well
Royalite No. 28

and in such other wells as the Board may, from time to time, direct or authorize.

(3) The injection of water

directly and personally supervising such practices, and of all directors, officers and shareholders of the corporation.

6. Any permit issued under this Article may be revoked, or its renewal withheld, by the Council for failure of the permit holder to observe any of the conditions set forth herein governing the issuance of a permit, or where the permit holder, or any of its officers, directors, shareholders or employees has or have been guilty of conduct that, in the judgment of the Council, is contrary to the best interests of the public or of the practice of veterinary medicine.

ALBERTA REGULATION 374/71

(Filed December 23, 1971)

THE PUBLIC HEALTH ACT

(O.C. 2157/71)

Approved and Ordered,
GRANT MacEWAN,
Lieutenant Governor. Edmonton, December 22, 1971.

The Executive Council has had under consideration the report of the Honourable the Minister of Health and Social Development, dated December 13, 1971, stating that:

Whereas the Provincial Board of Health has made a regulation in accordance with the Appendix attached hereto:

Therefore, upon the recommendation of the Honourable the Minister of Health and Social Development, the Executive Council advises that the Lieutenant Governor in Council, pursuant to section 7, subsection (1) of The Public Health Act, hereby approves the regulation in the Appendix attached hereto, being Regulation to Amend the Provincial Board of Health Regulations under The Public Health Act.

PETER LOUGHEED (Chairman).

PROVINCIAL BOARD OF HEALTH

I, P. B. Rose, Chairman of the Provincial Board of Health, hereby certify that the attached copy of a regulation made under section 7, subsection (1) of The Public Health Act, entitled Regulation to Amend the Provincial Board of Health Regulations Under The Public Health Act is a true copy of the regulation made by the Board on December 8, 1971.

PAT B. ROSE.

REGULATION TO AMEND THE PROVINCIAL BOARD OF HEALTH REGULATIONS UNDER THE PUBLIC HEALTH ACT

1. Alberta Regulation 572/57, as amended, is further amended as follows:

2. Division 25, Regulations Respecting the Protection of Persons

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ALBERTA REGULATION 375/71

(Filed December 23, 1971)

THE PUBLIC HEALTH ACT

(O.C. 2156/71)

Approved and Ordered,
GRANT MacEWAN,
Lieutenant Governor. Edmonton, December 22, 1971.

The Executive Council has had under consideration the report of the Honourable the Acting Minister of Health and Social Development, dated December 13, 1971, stating that:

Whereas the Provincial Board of Health has made regulations in accordance with the Appendix attached hereto:

Therefore, upon the recommendation of the Honourable the Acting Minister of Health and Social Development, the Executive Council advises that the Lieutenant Governor in Council, pursuant to section 7, subsection (1) of The Public Health Act, hereby approves the regulations in the Appendix attached hereto, being Provincial Board of Health Regulations Respecting the Protection of Persons from Fibrosis of the Lungs.

PETER LOUGHEED (Chairman).

THE PROVINCIAL BOARD OF HEALTH

I, P. B. Rose, Chairman of the Provincial Board of Health, hereby certify that the attached copy of regulations under section 7, subsection (1) of The Public Health Act, entitled Provincial Board of Health Regulations Respecting the Protection of Persons from Fibrosis of the Lungs, is a true copy of the regulations made by the Board on December 8, 1971.

PAT B. ROSE.

PROVINCIAL BOARD OF HEALTH REGULATIONS RESPECTING THE PROTECTION OF PERSONS FROM FIBROSIS OF THE LUNGS

(DIVISION 25)

Application

25-1-1 Every person who is engaged in any occupation where he is or may be exposed to the inhalation of any substance which may produce fibrosis of the lungs shall submit not less than once every two years to an examination to include a 14" x 17" chest X-ray and a pulmonary function test (to include FVC and FEV, 1.0 seconds).

25-1-2 Without limiting the generality of section 25-1-1, it shall be considered to apply to any person engaged in any of the occupations or groups of occupations listed hereafter:

(a) *Group 1 Occupations* — Including occupations involving exposure of the worker to:

(i) *Free Silica Dust*

Cement making,
Coal mining,
Concrete making and breaking,
Demolition,
Foundry work,
Glass making,
Gravel road maintenance,
Hard Rock mining,
Pottery and ceramics making,
Quarrying and stone dressing,
Rock and gravel crushing,
Sandblasting,
Steel manufacture,
Street sweeping,
Tunnelling,
and

(ii) *Carbonaceous Materials*

Coal,

and

(b) *Group 2 Occupations* — Including any or all occupations involving exposure of the worker to asbestos dust, asbestiform material or any compound containing asbestos particularly:

Asbestos processors,
Auto-body workers,
Construction workers,
Demolition workers,
Insulation workers,

and

(c) *Group 3 Occupations* — Including occupations which involve exposure of the worker to organic dusts and shall include the following:

Feed Mill operators,
Grain elevator operators,
Seed cleaning and processing plant operators,
Woodworkers.

Examinations, Certificates and Records

25-2-1 (1) Every examination required pursuant to these regulations shall be performed by an official or officials designated by the Provincial Board of Health to conduct such examinations.

(2) Any fee payable for an examination required by these regulations shall be paid by the employer and there shall be no charge to the person examined for the required examination.

25-2-2 (1) The official or officials conducting the required examination shall forward or cause to be forwarded the X-ray plate and

charts recording the Pulmonary Function test findings to the Director of the Division of Industrial Health Services of the Alberta Department of Health and Social Development.

(2) The Director of the Division of Industrial Health Services shall then issue the certificate required under section 25-2-3 when he is satisfied that the entire required examination has been completed.

25-2-3 (1) Every person who is examined under the provisions of sections 25-2-1 and 25-2-2 shall be issued with a certificate indicating that the examination required by these Provincial Board of Health Regulations has been carried out.

(2) The certificate authorized by subsection (1) shall be signed by the Director of the Division of Industrial Health Services, or by a physician duly authorized by the Provincial Board of Health.

(3) Any person receiving the certificate authorized by this section shall furnish the same to the manager, superintendent or other person in charge of the plant or activity employing him.

25-2-4 (1) The manager, superintendent or other person in charge of any plant or activity where any person or persons are engaged in any of the occupations referred to in section 25-1-1 shall obtain from every employee so engaged within one month of his commencing such employment the certificate referred to in section 25-2-3 indicating that the required examination has been carried out.

(2) The certificate required by section 25-2-3 shall be reviewed at intervals which shall be not more than two years apart, except as provided in subsection (3) hereafter.

(3) Where due to exigencies which may arise, it may not be practicable for an official designated by the Provincial Board of Health to conduct the examination required by these regulations, the Provincial Board of Health may direct that the interval between such examinations be extended beyond the two year period for such further period as the said Board in its discretion may direct.

25-2-5 (1) Any person who has been issued a certificate under section 25-2-3 indicating that the examination required by these regulations has been carried out and who has furnished such certificate to the manager, superintendent or other person in charge of the plant or activity and who subsequently severs his employment shall be entitled on request to have such certificate returned to him.

(2) If a person who obtained a certificate under the provisions of subsection (1) is engaged within two years of the date of the certificate in any occupation listed in section 25-1-1 he may furnish such certificate to the manager, superintendent or other person in charge of the plant or activity employing him and in such case the certificate shall continue to be valid for all purposes of these regulations for a period of two years from the date of the certificate.

25-2-6 The certificate required by section 25-2-4 and furnished under section 25-2-5 shall be kept on file by the manager, superintendent or other person in charge and [illegible]

25-2-7 The manager, superintendent or other person in charge shall not permit any person to continue in employment contrary to the provisions of sections 25-2-4 and 25-2-5.

Time for Examinations

25-3-1 The manager, superintendent or other person in charge of the plant or activity shall permit any person who is engaged in any occupation listed in section 25-1-1 to undergo the examination required by these regulations during working hours under such arrangements as may be directed by the official designated by the Provincial Board of Health to conduct such examination and the manager, superintendent or other person in charge shall ensure that any person examined under the provisions of this division receives pay for the time necessary for this examination in the same amount as he would otherwise have received for that time.

ALBERTA REGULATION 376/71

(Filed December 23, 1971)

THE AGRICULTURAL SOCIETIES ACT

(O.C. 2160/71)

Approved and Ordered,
GRANT MacEWAN,
Lieutenant Governor. Edmonton, December 22, 1971.

Upon the recommendation of the Honourable the Minister of Agriculture, dated December 13, 1971, the Executive Council advises that the Lieutenant Governor in Council, pursuant to section 37 of The Agricultural Societies Act, hereby makes regulations in accordance with the Appendix attached hereto, being Regulations to Amend Regulations Governing Capital Grants.

PETER LOUGHEED (Chairman).

REGULATIONS TO AMEND REGULATIONS GOVERNING CAPITAL GRANTS

1. Alberta Regulation 363/71, being Regulations Governing Capital Grants, is hereby amended.

2. The following section is added, immediately after section 6

"Alberta Regulation 78/59, being Regulations Respecting The Payment of Capital Grants To "B" Class Fairs, is hereby rescinded."

ALBERTA REGULATION 377/71

(Filed December 23, 1971)

THE LIQUOR CONTROL ACT

(O.C. 2162/71)

Approved and Ordered,
GRANT MacEWAN,
Lieutenant Governor. Edmonton, December 22, 1971.

The Executive Council has had under consideration the report of

Whereas The Alberta Liquor Control Board has, by an Order dated December 13, 1971, made the regulations in the appendix attached hereto:

Therefore, upon the recommendation of the Honourable the Attorney General, the Executive Council advises that the Lieutenant Governor in Council hereby approves the regulations in accordance with the appendix attached hereto, being Regulations to amend Regulations under The Liquor Control Act.

PETER LOUGHEED (Chairman).

BOARD ORDER NO. ADM-1397

An Order making Regulations to amend Regulations under The Liquor Control Act, filed as Alberta Regulation 164/71.

Whereas pursuant to the provisions of section 15 of The Liquor Control Act, the board may, with the approval of the Lieutenant Governor in Council, make such regulations as to it seem necessary for the carrying out of The Liquor Control Act and The Liquor Licensing Act, and for the efficient administration thereof;

And whereas the board has made regulations thereunder, which are attached as an appendix hereto;

Therefore it is ordered that Regulations be made in accordance with the appendix attached hereto, being Regulations to amend Regulations under The Liquor Control Act.

Dated at the City of Edmonton, in the Province of Alberta, this thirteenth day of December, 1971.

THE ALBERTA LIQUOR CONTROL BOARD,
A. D. ELLIOTT (Chairman).

REGULATIONS TO AMEND THE REGULATIONS UNDER THE LIQUOR CONTROL ACT

1. Alberta Regulation 164/71 is hereby amended.

2. Section 30 is struck out and the following is substituted:

30. Unless otherwise authorized by the Board, the maximum selling price charged by the holder of a resale special permit may not exceed

(a) Liquor — fifty cents per ounce
(b) Alberta beer of an alcoholic strength not over 3.9% of alcohol by volume — thirty cents per bottle
(c) Alberta beer of an alcoholic strength not over 5.5% of alcohol by volume — thirty-five cents per bottle
(d) Imported beer — fifteen cents per bottle over the purchase price paid to the Board
(e) Wine — 50% over the

APPENDIX

OCCUPATIONAL HEALTH AND SAFETY ACT

Chemical Hazards Regulation

1(1) In this regulation

(a) "8 hour Occupational Exposure Limit" means the time-weighted average concentration of an airborne substance listed in Schedule A for an 8 hour period;

(b) "15 minute Occupational Exposure Limit" means the time-weighted average concentration of an airborne substance listed in Schedule A for a 15 minute period;

(c) "ceiling Occupational Exposure Limit" means the maximum concentration of an airborne substance listed in Schedule A;

(d) "mg/m^3" means milligrams of substance per cubic metre of air measured at standard conditions of 25 degrees C and 100 kiloPascals;

(e) "ppm" means parts of vapor or gas by volume per million parts of contaminated air by volume;

(f) "R" means registered trade mark;

(g) "respirable mass" means that weight of the total airborne particulate which can be inhaled and deposited in the lower respiratory tract;

(h) "Skin" when it appears in conjunction with a substance in Schedule A means the substance can be absorbed through the intact skin.

(2) In measuring the Occupational Exposure Limit for a particular substance, an employer shall bedeemed not to have contravened this regulation respecting the prescribed Occupational Exposure Limit if he complies with the Occupational Exposure Limit requirements with respect to either the ppm or the mg/m^3 measure.

2(1) An employer shall ensure that a worker is not exposed by inhalation to any substance listed in Table 1 or Table 2 of Schedule A in excess of the prescribed Occupational Exposure Limit.

(2) For the purposes of calculating the 15-minute Occupational Exposure Limit in Table 1 and Table 2 of Schedule A

(a) not more than 4 15-minute periods shall be permitted per shift, and

(b) there must be at least 60 minutes between each period referred to in clause (a).

3 An employer shall take all reasonable steps to ensure that where a worker is exposed to more than one substance listed in Table 1 or 2 of Schedule A in the same shift, and where the toxicological effects of those substances are additive, the value of D in the formula below does not exceed 1,

$$D = \frac{C_1}{T_1} + \frac{C_2}{T_2} + \ldots + \frac{C_n}{T_n}$$

where C_1, C_2 ... C_n refer to the actual airborne concentrations of contaminants 1, 2, ... n, and T_1, T_2 ... T_n are their respective Occupational Exposure Limit.

4 An employer shall take all reasonable steps to ensure that where a substance listed in Schedule B contaminates the air at a work site the contamination does not present a fire or explosion hazard and does not reduce the available oxygen content of the air below a partial pressure of 18 kiloPascals.

5 An employer shall take all reasonable steps to ensure that where a worker is exposed to any substance listed in Table 1 or Table 2 of Schedule A in excess of the prescribed Occupational Exposure Limit

(a) the worker is immediately protected from further exposure in excess of the prescribed Occupational Exposure Limit,

(b) the source of contamination is immediately identified and steps are forthwith taken to control the contamination, and

(c) the work site safety committee, if any, is informed in writing within 24 hours of the worker exposure and of the steps taken to control the contamination.

6 An employer shall notify the Director of Occupational Hygiene in writing within 14 days after the date any substance or process listed in Schedule C is brought to, stored or used at the work site, and shall advise him of the name of the substance or process and the protection used by the employer to control worker exposure to the substance or process.

7 An employer shall ensure that any substance or process listed in Schedule C is controlled in a manner that is approved by the Director of Occupational Hygiene as adequately protecting the health of workers at the work site.

8 Where a worker is or may be exposed to any substance listed in Schedule A, B or C the employer shall ensure that each such worker

(a) is trained in and utilizes training in procedures that minimize the worker's exposure to the substance,

(b) is instructed in the purpose, proper use and limitations of any protective equipment provided, and

(c) is instructed regarding the health hazards associated with exposure to that substance.

9 For the purposes of section 2, an employer shall ensure that no worker is exposed to an airborne substance in excess of the Occupational Exposure Limits set out in Tables 1 and 2 of Schedule A by first taking all reasonable steps to institute engineering, work practice or administrative controls, and, if such reasonable steps are not effective to keep exposure under those limits, then by supplying protective equipment to the

worker in accordance with this regulation and any other regulations under the Act.

10 Where a respiratory protective device is used to control worker exposure to a substance listed in Schedule A, B, or C the employer shall

(a) provide and ensure that each exposed worker wears an appropriate, correctly fitted device,

(b) ensure that each worker provided with a device is instructed in the purpose, proper use and limitations of the device,

(c) ensure that each device provided to a worker is stored in a manner which prevents contamination of it, and

(d) ensure that each device provided to a worker is either properly maintained or replaced so that filtering effectiveness is not impaired, and that each device to be reused is regularly cleaned.

11 An employer shall provide and ensure that each worker wears appropriate skin and eye protection where

(a) the worker is working with any substance that may damage the skin or eyes on contact, or

(b) the worker is working with any substance listed in Schedule A with a "Skin" notation where that substance is in a state that may be absorbed through intact skin.

12 A worker shall

(a) wear any respiratory protective device, skin protection, eye protection or protective clothing when it is provided by an employer,

(b) follow procedures set by the employer under section 8(a), and

(c) participate in any instruction provided under section 8(b) or (c).

13 The substances listed in Tables 1 and 2 of Schedule D are designated substances for the purposes of section 24 of the Act.

14 The Provincial Board of Health Regulations Respecting Occupational Health (Alta. Reg. 298/72) are repealed.

15 If a provision in this regulation conflicts with a provision in Alta. Reg. 287/76 or 270/76, the provision in this regulation prevails.

16 This regulation comes into force on March 1, 1982.

SCHEDULE A

TABLE 1

SUBSTANCE	Prescribed 8 hr. Occupational Exposure Limit		Prescribed 15 min Occupational Exposure Limit		Prescribed CEILING Occupational Exposure Limit	
	ppm	mg/m³	ppm	mg/m³	ppm	mg/m³
Abate	---	10	---	20	---	---
Acetaldehyde	100	180	150	270	---	---
Acetic acid	10	26	15	39	---	---
Acetic anhydride	---	---	---	---	5	21
Acetone	1000	2370	1250	2970	---	---
Acetonitrile - Skin	40	67	60	100	---	---
Acetylene dichloride						
(1,2-Dichloroethylene)	200	793	250	995	---	---
Acetylene tetrabromide	1	14	1.5	21	---	---
Acrolein	0.1	0.23	0.3	0.69	---	---
Acrylamide - Skin	---	0.3	---	0.6	---	---
Acrylic acid	10	30	20	60	---	---
Acrylonitrile - Skin	2	4.3	4	8.6	---	---
Aldrin - Skin	---	0.25	---	0.75	---	---
Allyl alcohol - Skin	2	4.7	4	9.5	---	---
Allyl chloride	1	3.2	2	6.3	---	---
Allyl glycidyl ether (AGE)-Skin	5	23	10	47	---	---
Allyl propyl disulfide	2	12	3	18	---	---
Aluminum metal & oxide	---	10	---	20	---	---
Aluminum pyro powders	---	5	---	10	---	---
Aluminum welding fumes	---	5	---	10	---	---
Aluminum soluble salts	---	2	---	4	---	---
Aluminum, alkyls	---	2	---	4	---	---
2-Aminoethanol (Ethanolamine)	3	7.5	6	15	---	---
2-Aminopyridine	0.5	1.9	2	7.7	---	---
Ammonia	25	17	35	24	---	---
Ammonium chloride - fume	---	10	---	20	---	---

SCHEDULE A

TABLE 2

	Prescribed 8 hr. Occupational Exposure Limit	
	Respirable Mass mg/m^3	Total Mass mg/m^3
Silica (SiO_2)		
Amorphous	2	5
Cristobalite	0.05	0.15
Fused Silica	0.1	0.3
Quartz	0.1	0.3
Silica Flour	0.05	0.15
Tridymite	0.05	0.15
Tripoli	0.1	0.3
Aluminum oxide (Al_2O_3)	5	10
Asbestos	see footnote (a)	
Calcium carbonate	5	10
Calcium silicate	5	10
Cellulose (paper fiber)	5	10
Coal Dust	see footnote (b)	
Emery	5	10
Fibrous Glass	see footnote (c)	
Graphite (Synthetic)	5	10
Gypsum	5	10
Kaolin	5	10
Limestone	5	10
Marble	5	10
Magnesite	5	10
Mica	3	6
Mineral Wool Fibre	see footnote (c)	
Nuisance Particulate	5	10
Pentaerythritol	5	10
Perlite	5	10
Plaster of Paris	5	10
Portland Cement	5	10
Rouge	5	10
Silicon	5	10
Silicon carbide	5	10
Soapstone	3	6
Starch	5	10
Sucrose	5	10
Talc (fibrous)	see footnote (d)	
Talc (nonasbestiform)	3	6
Tin oxide	5	10
Titanium dioxide	5	10
Zinc stearate	5	10
Zinc oxide dust	5	10

(a) asbestos

(i) for asbestos fibre, except crocidolite, amosite and tremolite, the 8 hour Occupational Exposure Limit is 2 fibres greater than 5 micrometers in length per cm^3 of air;

the 15 minute Occupational Exposure Limit is 10 fibres greater than 5 micrometers in length per cm^3 of air;

(ii) for crocidolite fibre, the 8 hour Occupational Exposure Limit is 0.2 fibres greater than 5 micrometers in length per cm^3 of air;

the 15 minute Occupational Exposure Limit is 1 fibre greater than 5 micrometers in length per cm^3 of air;

(iii) for amosite and tremolite fibre, the 8 hour Occupational Exposure Limit is 0.5 fibres greater than 5 micrometers in length per cm^3 of air;

the 15 minute Occupational Exposure Limit is 2.5 fibres greater than 5 micrometers in length per cm^3 of air;

(b) coal dust

(i) in underground coal mines, the 8 hour Occupational Exposure Limit is 5 mg/m^3 (respirable mass);

(ii) in surface coal mines and coal processing plants, the 8 hour Occupational Exposure Limit is 2 mg/m^3 (respirable mass);

(c) fibrous glass or mineral wool fibre

(i) for fibrous glass or mineral wool fibre, the 8 hour Occupational Exposure Limit is 3 fibres per cm^3 of air;

(ii) fibres included in this count are those having a diameter equal to or less than 3.5 micrometers and a length equal to or greater than 10 micrometers;

(iii) the 8 hour Occupational Exposure Limit is 5 mg/m^3 (total mass);

(d) talc (fibrous)

(i) for fibrous talc, the 8 hour Occupational Exposure Limit is 2 fibres greater than 5 micrometers in length per cm^3 of air;

(ii) the 15 minute Occupational Exposure Limit is 10 fibres greater than 5 micrometers in length per cm^3 of air.

SCHEDULE B

Acetylene
Argon
Ethane
Ethylene
Helium
Hydrogen
Methane
Neon
Nitrogen
Propane
Propylene

SCHEDULE C

Acrylonitrile
4-Aminodiphenyl
3-Amino-1,2,4-triazole
Antimony trioxide production
Arsenic trioxide production
Asbestos
Benzene
Benzidine production
Benzo(a)pyrene
Beryllium
Cadmium oxide production
bis-Chloromethyl ether
Chromates - water insoluble Cr VI compounds
Chromite ore processing
Chrysene
Coal tar pitch volatiles
1,2-Dibromoethane (Ethylene dibromide)
3,3'-Dichlorobenzidine
Dimethyl carbamyl chloride
1,1'-Dimethyl hydrazine
Dimethyl sulfate
Ethylene dibromide
Hexachlorobutadiene
Hexamethyl phosphoramide
Hydrazine
Lead Chromate
4,4'-Methylene bis(2-chloroaniline)
Methyl hydrazine
Methyl iodide
beta-Naphthylamine
Nickel sulfide roasting
4-Nitrodiphenyl
2-Nitropropane
N-Nitrosodimethylamine (Dimethylnitrosoamine)
N-Phenyl-beta-naphthylamine
Propane sultone
beta-Propiolactone
o-Toluidine
Vinyl bromide
Vinyl chloride
Vinyl cyclohexene dioxide
Zinc chromate

ALBERTA REGULATION 9/82

(Filed on January 7, 1982)

OCCUPATIONAL HEALTH AND SAFETY ACT

(O.C. 14/82)

Approved and Ordered,
F. LYNCH-STAUNTON,
Lieutenant Governor.

Edmonton, January 6, 1982

Upon the recommendation of the Honourable Mr. Diachuk, the Lieutenant Governor in Council, pursuant to section 31(1) of the Occupational Health and Safety Act, makes the regulation in the attached Appendix, being the Silica Regulation.

PETER LOUGHEED (Chairman)

APPENDIX

OCCUPATIONAL HEALTH AND SAFETY ACT

Silica Regulation

1 In this regulation

(a) "FEV$_1$" means forced expiratory volume in 1.0 second;

(b) "FVC" means forced vital capacity;

(c) "pulmonary function technician" means a person who has passed a pulmonary function technician course approved by the Director of Medical Services, or has been approved by the Director as having the equivalent of an approved pulmonary function technician course and who, in either case, has passed a requalification examination when requested by the Director to take one;

(d) "silica" means silicon dioxide, hydrated silicon dioxide, fused silica, silica flour, quartz, cristobalite and tridymite.

2 This regulation does not apply to a person to whom the Provincial Board of Health Regulations Respecting the Protection of Persons From Fibrosis of the Lungs (Alta. Reg. 375/71) apply.

3 An employer shall

(a) keep work areas clear of unnecessary accumulations of silica dust;

(b) wet down the silica dust where practicable;

(c) ensure that any cleaning of a work area where silica dust is present is done by wet sweeping or by use of a vacuum cleaner equipped with a filter that is adequate to prevent silica dust from being discharged with the air that is discharged from the vacuum cleaner.

4 An employer shall provide a manual of safe practice procedures for each operation that could result in a worker's exposure to airborne silica, and ensure that copies of the manual of safe practice procedures and copies of this regulation are readily available to each worker.

5 Sections 3 and 4 do not apply to work areas in which the employer establishes to the satisfaction of the Director of Occupational Hygiene that worker exposure to airborne silica is 50% or less of the 8-hour Occupational Exposure Limit set out in the Chemical Hazards Regulation.

6(1) For the purposes of this section "exposed worker" means a worker who, for at least 30 days in a 12-month period, will likely be exposed to airborne silica in excess of 50% of the 8-hour Occupational Exposure Limit set out in the Chemical Hazards Regulation.

(2) The employer of an exposed worker shall ensure that the worker undergoes a medical assessment within 30 days after his first exposure and not later than every 24 months after the date of that assessment for as long as the worker continues to be an exposed worker and, for the purposes of determining whether a worker is an exposed worker under this subsection, the first 12-month period commences on the date of the worker's first exposure to airborne silica.

(3) If, on the coming into force of this regulation an exposed worker is being examined in accordance with Alta. Reg. 375/71, the employer shall ensure that the worker undergoes a medical assessment not later than 24 months after his last examination under that regulation and not later than every 24 months after the date of that assessment for as long as the worker continues to be an exposed worker and, for the purposes of determining whether a worker is an exposed worker under this subsection, the first 12-month period commences on the date this regulation comes into force.

7 A medical assessment shall consist of

(a) a P.A. chest x-ray of the worker on a 35 cm by 43 cm plate,

(b) a pulmonary function test including the spirogram, FEV_1 and FVC, conducted by a pulmonary function technician,

(c) an assessment of the worker's ability to wear a respiratory protective device,

(d) a written history specifying the worker's

(i) occupational exposure to industrial dust,

(ii) personal and family incidence of pulmonary tuberculosis,

(iii) respiratory symptoms including dysponea, cough, sputum production, wheezing or chest tightness,

(iv) incidence of chronic bronchitis, emphysema, asthma and other chronic lung disease, and

(v) smoking history.

8(1) An employer shall bear the cost of providing medical assessments under this regulation.

(2) A person who administers an x-ray or pulmonary function test or prepares an assessment or written history under section 7 shall, within 30 days thereafter forward to the Director of Medical Services the x-ray plate, the results of the pulmonary function test, a copy of the assessment or a copy of the written history as the case may be.

9 A worker shall participate in a medical assessment provided pursuant to this regulation.

10 Silicosis and pneumoconiosis are designated as notifiable diseases for the purpose of section 17 of the Act.

11 If a provision in this regulation conflicts with a provision in Alta. Reg. 267/76 or 271/76, the provision in this regulation prevails.

12 *The Provincial Board of Health Regulations Respecting the Protection of Persons From Fibrosis of the Lungs, (Alta. Reg. 375/71) are amended by repealing sections 25-1-1 and 25-1-2 and substituting the following:*

25-1-1 A person who is engaged in coal mining and is or may be exposed to the inhalation of free silica dust or carbonaceous materials shall submit not less than once every 2 years to an examination, to include a 14" X 17" chest x-ray and a pulmonary function test (to include FVC and FEV, 1.0 seconds).

13 This regulation comes into force on March 1 1982

ALBERTA REGULATION 6/82

(Filed on January 7, 1982)

PUBLIC TRUSTEE ACT

(O.C. 11/82)

Approved and Ordered,
F. LYNCH-STAUNTON,
Lieutenant Governor.

Edmonton, January 6, 1982

Upon the recommendation of the Honourable the Attorney General, the Lieutenant Governor in Council, pursuant to section 26 of the Public Trustee Act, makes the regulation in the attached Appendix, being the Public Trustee Common Fund Interest Rate Regulation.

PETER LOUGHEED (Chairman)

APPENDIX

PUBLIC TRUSTEE ACT

Public Trustee Common Fund Interest Rate Regulation

1 On and after November 1, 1981 the interest payable in respect of estates, the money of which forms the common fund, shall be at the rate of 13% per annum.

2 The Public Trustee Common Fund Interest Rate Regulation (Alta. Reg. 146/80) is repealed.

ALBERTA REGULATION 7/82

(Filed on January 7, 1982)

OCCUPATIONAL HEALTH AND SAFETY ACT

(O.C. 12/82)

Approved and Ordered,
F. LYNCH-STAUNTON,
Lieutenant Governor.

Edmonton, January 6, 1982

Upon the recommendation of the Honourable Mr. Diachuk, the Lieutenant Governor in Council, pursuant to section 31(1) of the Occupational Health and Safety Act, makes the regulation in the attached Appendix, being the Asbestos Regulation.

PETER LOUGHEED (Chairman)

APPENDIX

OCCUPATIONAL HEALTH AND SAFETY ACT

Asbestos Regulation

1 In this regulation

(a) "asbestos" means chrystotile, crocidolite, amosite, tremolite, anthophyllite and actinolite when in their fibrous form;

(b) "FEV_1" means forced expiratory volume in 1.0 second;

(c) "FVC" means forced vital capacity;

(d) "pulmonary function technician" means a person who has passed a pulmonary function technician course approved by the Director of Medical Services, or has been approved by the Director as having the equivalent of an approved pulmonary function technician course and who, in either case, has passed a requalification examination when requested by the Director to take one;

(e) "restricted area" means an area of a work site in which there is a reasonable potential for worker exposure to airborne asbestos in an amount equal to or greater than 25% of the 8-hour Occupational Exposure Limit in the Chemical Hazards Regulation.

2 An employer shall

(a) keep his work site clear of unnecessary accumulations of asbestos waste;

(b) wet the asbestos for handling where practicable;

(c) ensure that any cleaning of a restricted area is done by wet sweeping or by use of a vacuum cleaner equipped with a filter that is adequate to prevent asbestos fibre from being discharged with the air that is discharged from the vacuum cleaner;

(d) ensure that containers of asbestos products are clearly labelled to indicate the contents and carcinogenic hazard, with a warning that the dust should not be breathed;

(e) ensure that all asbestos waste is kept, conveyed and disposed of in sealed containers that are impervious to asbestos and are clearly labelled to indicate the contents and carcinogenic hazard, with a warning that the dust should not be breathed.

3 In any case where a worker is employed in connection with the demolition of structures containing asbestos or in connection with removal of insulation containing asbestos, the worker's employer shall provide to the worker and ensure that he wears an appropriate, correctly fitted respiratory protective device.

4 An employer shall

(a) provide a manual of safe practice procedures for each operation that could result in a worker's exposure to asbestos,

(b) ensure that copies of the manual of safe practice procedures and copies of this regulation are readily available to each worker,

(c) ensure that each potentially exposed worker is warned in the hazard of smoking as it relates to asbestos exposure,

(d) restrict access to a restricted area to persons he authorizes and any other persons duly authorized by law,

(e) post in a conspicuous place at the entrances to or on the periphery of a restricted area, signs that clearly indicate

(i) that asbestos is present in the area,

(ii) that access to the area is prohibited, except to authorized personnel, and

(iii) that drinking, eating and smoking are prohibited in the area,

(f) provide and ensure that a worker wears suitable protective clothing in a restricted area,

(g) provide and ensure that a worker utilizes a suitable storage area for protective clothing worn in a restricted area,

(h) ensure that reusable protective clothing worn in a restricted area is laundered when necessary and in any event not less frequently than every 3 days of use,

(i) ensure that protective clothing to be laundered is transported from a restricted area in sealed containers that are clearly labelled to indicate the contents and carcinogenic hazard with a warning that the dust should not be breathed,

(j) ensure that used disposable protective clothing is treated as asbestos waste, and

(k) ensure that compressed air is not used to clean protective clothing.

5 No person shall drink, eat or smoke in a restricted area.

6(1) For the purposes of this section "exposed worker" means a worker who, for at least 30 days in a 12-month period, will likely be exposed to airborne asbestos in an amount equal to or greater than 25% of the 8-hour Occupational Exposure Limit in the Chemical Hazards Regulation.

(2) The employer of an exposed worker shall ensure that the worker undergoes a medical assessment within 30 days after his first exposure and

(a) not later than every 24 months after the date of that assessment, for the first 10 12-month periods if the worker continues to be an exposed worker for those periods, and

(b) not later than every 12 months after the date of the last assessment conducted under clause (a), for as long as the worker continues to be an exposed worker,

and for the purposes of determining whether a worker is an exposed worker under this subsection the first 12-month period commences on the date of the worker's first exposure to airborne asbestos.

(3) Subject to subsection (4), if on the coming into force of this regulation an exposed worker is being examined in accordance with Alta. Reg. 375/71, the employer shall ensure that the worker undergoes a medical assessment not later than 24 months after his last examination under that regulation and

(a) not later than every 24 months after the date of that assessment, for the first 10 12-month periods if the worker continues to be an exposed worker for those periods, and

(b) not later than every 12 months after the date of the last assessment conducted under clause (a), for as long as the worker continues to be an exposed worker,

and for the purposes of determining whether a worker is an exposed worker under this subsection the first 12-month period commences on the date this regulation comes into force.

(4) In the case of a worker who is, on the coming into force of this regulation, being examined in accordance with Alta. Reg. 375/71, each 12-month period preceding the coming into force of this regulation in respect of which he was being so examined shall be considered to be one of the 10 12-month periods for the purposes of subsection 3(a).

(5) A medical assessment shall consist of

(a) a P.A. chest x-ray on a 35 cm by 43 cm plate,

(b) a pulmonary function test, including the spirogram, FEV_1 and FVC, all conducted by a pulmonary function technician,

(c) an assessment of the worker's ability to wear a respiratory protective device, and

(d) a written history specifying the worker's

(i) occupational exposure to industrial dust and carcinogens,

(ii) respiratory symptoms including dyspnoea, cough, sputum production, wheezing or chest tightness,

(iii) incidence of asthma, chronic bronchitis, emphysema, lung cancer, or other chronic lung disease, and

(iv) smoking history.

7(1) An employer shall bear the cost of providing medical assessments under this regulation.

(2) A person who administers an x-ray or pulmonary function test or prepares an assessment or written history under section 6(5) shall, within 30 days thereafter forward to the Director of Medical Services the x-ray plate, the results of the pulmonary function test, a copy of the assessment or a copy of the written history, as the case may be.

8 A worker shall participate in a medical assessment provided pursuant to this regulation.

9 Asbestosis, mesothelioma and asbestos induced lung, laryngeal and gastro-intestinal cancer are designated as notifiable diseases for the purpose of section 17 of the Act.

10 If a provision in this regulation conflicts with a provision in Alta. Reg. 267/76 or 271/76, the provision in this regulation prevails.

11 This regulation comes into force on March 1, 1982.

ALBERTA REGULATION 8/82

(Filed on January 7, 1982)

OCCUPATIONAL HEALTH AND SAFETY ACT

(O.C. 13/82)

Approved and Ordered,
F. LYNCH-STAUNTON,
Lieutenant Governor.

Edmonton, January 6, 1982

Upon the recommendation of the Honourable Mr. Diachuk, the Lieutenant Governor in Council, pursuant to section 31(1) of the Occupational Health and Safety Act, makes the regulation in the attached Appendix, being the Chemical Hazards Regulation.

PETER LOUGHEED (Chairman)

APPENDIX

OCCUPATIONAL HEALTH AND SAFETY ACT

Chemical Hazards Amendment Regulation

1 The Chemical Hazards Regulation (Alta. Reg. 8/82) is amended by this regulation.

2 Section 1 is amended

(a) in subsection (1) by adding the following after clause (c):

(c.1) "coal dust" means a dust

(i) resulting from the mining, transport or processing of coal,

(ii) of carboniferous or mixed mineralogical composition, and

(iii) containing 10% or less of free silica calculated by weight;

(b) in subsection (2) by striking out "shall bedeemed" and substituting "shall be deemed".

3 Tables 1 and 2 of Schedule A are repealed and the attached Tables 1 and 2 are substituted.

4 Schedule C is repealed and the attached Schedule C is substituted.

5 Tables 1 and 2 of Schedule D are repealed and the attached Tables 1 and 2 are substituted.

SCHEDULE A

TABLE 1

SUBSTANCE	Prescribed 8 hr. Occupational Exposure Limit		Prescribed 15 min Occupational Exposure Limit		Prescribed CEILING Occupational Exposure Limit	
	ppm	mg/m^3	ppm	mg/m^3	ppm	mg/m^3
Abate	---	10	---	20	---	---
Acetaldehyde	100	180	150	270	---	---
Acetic acid	10	26	15	39	---	---
Acetic anhydride	---	---	---	---	5	21
Acetone	750	1782	1000	2375	---	---
Acetonitrile - Skin	40	67	60	100	---	---
Acetylene (Schedule B)	---	---	---	---	---	---
Acetylene dichloride (1,2-Dichloroethylene)	200	795	250	995	---	---
Acetylene tetrabromide	1	14	1.5	21	---	---
Acrolein	0.1	0.23	0.3	0.69	---	---
Acrylamide - Skin	---	0.3	---	0.6	---	---
Acrylic acid	10	30	20	60	---	---
Acrylonitrile - Skin (Schedule C)	2	4.3	4	8.6	---	---
Aldrin - Skin	---	0.25	---	0.75	---	---
Allyl alcohol - Skin	2	4.7	4	9.5	---	---
Allyl chloride	1	3.2	2	6.3	---	---
Allyl glycidyl ether (AGE)-Skin	5	23	10	47	---	---
Allyl propyl disulfide	2	12	3	18	---	---
Aluminum metal & oxide	---	10	---	20	---	---
Aluminum pyro powders	---	5	---	10	---	---
Aluminum welding fumes	---	5	---	10	---	---
Aluminum soluble salts	---	2	---	4	---	---
Aluminum, alkyls	---	2	---	4	---	---
4-Aminodiphenyl (Schedule C)	---	---	---	---	---	---
2-Aminoethanol (Ethanolamine)	3	7.5	6	15	---	---
2-Aminopyridine	0.5	1.9	2	7.7	---	---
3-Amino-1,2,4-triazole (Schedule C)	---	---	---	---	---	---

SCHEDULE A

TABLE 2

	Prescribed 8 hr. Occupational Exposure Limit	
	Respirable Mass mg/m^3	Total Mass mg/m^3
Silica (SiO_2)		
Amorphous	2	5
Cristobalite	0.05	0.15
Fused Silica	0.1	0.3
Quartz	0.1	0.3
Silica Flour	0.05	0.15
Tridymite	0.05	0.15
Tripoli	0.1	0.3
Aluminum oxide (Al_2O_3)	5	10
Asbestos (Schedule C)	see footnote (a)	
Calcium carbonate	5	10
Calcium silicate	5	10
Cellulose (paper fiber)	5	10
Coal Dust	see footnote (b)	
Emery	5	10
Fibrous Glass	see footnote (c)	
Graphite (Synthetic)	5	10
Gypsum	5	10
Kaolin	5	10
Limestone	5	10
Marble	5	10
Magnesite	5	10
Mica	3	6
Mineral Wool Fibre	see footnote (c)	
Nuisance Particulate	5	10
Pentaerythritol	5	10
Perlite	5	10
Plaster of Paris	5	10
Portland Cement	5	10
Rouge	5	10
Silicon	5	10
Silicon carbide	5	10
Soapstone	3	6
Starch	5	10
Sucrose	5	10
Talc (fibrous)	see footnote (d)	
Talc (nonasbestiform)	3	6
Tin oxide	5	10
Titanium dioxide	5	10
Zinc stearate	5	10
Zinc oxide dust	5	10

(a) asbestos

(i) for asbestos fibre, except crocidolite, amosite and tremolite, the 8 hour Occupational Exposure Limit is 2 fibres greater than 5 micrometers in length per cm^3 of air;

the 15 minute Occupational Exposure Limit is 10 fibres greater than 5 micrometers in length per cm^3 of air;

(ii) for crocidolite fibre, the 8 hour Occupational Exposure Limit is 0.2 fibres greater than 5 micrometers in length per cm^3 of air;

the 15 minute Occupational Exposure Limit is 1 fibre greater than 5 micrometers in length per cm^3 of air;

(iii) for amosite and tremolite fibre, the 8 hour Occupational Exposure Limit is 0.5 fibres greater than 5 micrometers in length per cm^3 of air;

the 15 minute Occupational Exposure Limit is 2.5 fibres greater than 5 micrometers in length per cm^3 of air;

(b) coal dust

(i) the 8 hour Occupational Exposure Limit is 2 mg/m^3 (respirable mass) average for all operations in the mine;

(ii) the 8 hour Occupational Exposure Limit is 4 mg/m^3 (respirable mass) in high risk operations;

(c) fibrous glass or mineral wool fibre

(i) for fibrous glass or mineral wool fibre, the 8 hour Occupational Exposure Limit is 3 fibres per cm^3 of air;

(ii) fibres included in this count are those having a diameter equal to or less than 3.5 micrometers and a length equal to or greater than 10 micrometers;

(iii) the 8 hour Occupational Exposure Limit is 5 mg/m^3 (total mass);

(d) talc (fibrous)

(i) for fibrous talc, the 8 hour Occupational Exposure Limit is 2 fibres greater than 5 micrometers in length per cm^3 of air;

(ii) the 15 minute Occupational Exposure Limit is 10 fibres greater than 5 micrometers in length per cm^3 of air.

SCHEDULE C

Acrylonitrile
4-Aminodiphenyl
3-Amino-1,2,4-triazole
Antimony trioxide production
Arsenic trioxide production
Asbestos
Benzene
Benzidine
Benzo(a)pyrene
Beryllium
Cadmium oxide production
Chloromethyl methyl ether
bis-Chloromethyl ether
Chromates - water insoluble Cr VI compounds
Chromite ore processing

Ethylene dibromide
Hexachlorobutadiene
Hexamethyl phosphoramide
Hydrazine
Lead Chromate
4,4'-Methylene bis(2-chloroaniline)
Methyl hydrazine
Methyl iodide
beta-Naphthylamine
Nickel sulfide roasting
4-Nitrodiphenyl

2-Nitropropane
N-Nitrosodimethylamine (Dimethylnitrosoamine)
N-Phenyl-beta-naphthylamine
Phenyl hydrazine

Trichloroethylene

Trichlorofluoromethane

Trichloronaphthalene

Trichloronitromethane (Chloropicrin)

1,2,3-Trichloropropane

1,1,2-Trichloro-1,2,2-trifluoroethane

Tricyclohexyltin hydroxide (Plictran^{R})

Triethylamine

Trifluoromonobromomethane

Trimellitic anhydride (TMA)

Trimethyl benzene

Trimethyl phosphite

2,4,6-Trinitrophenol (Picric acid)

2,4,6-Trinitrophenylmethylnitramine (Tetryl)

2,4,6-Trinitrotoluene (TNT)

Triorthocresyl phosphate

Triphenyl amine

Triphenyl phosphate

Tungsten and compounds

Turpentine

Uranium (natural) and compounds

Valeraldehyde

Vanadium (V_2O_5)

Vinyl acetate

Vinyl benzene (Styrene)

Vinyl cyanide (Acrylonitrile)

Vinylidene chloride

Vinyl toluene

VM & P Naphtha

Warfarin

Xylene (all isomers)

m-xylene alpha, alpha'-diamine

Xylidine

Yttrium

Zinc chloride

Zinc oxide

Zirconium compounds

ALBERTA REGULATION 243/83

(Filed on July 7, 1983)

OCCUPATIONAL HEALTH AND SAFETY ACT

(O.C. 567/83)

Approved and Ordered,
F. LYNCH-STAUNTON,
Lieutenant Governor,

Edmonton, July 6, 1983

Upon the recommendation of the Honourable the Minister responsible for Workers' Health, Safety and Compensation, the Lieutenant Governor in Council, pursuant to section 31(1) of the Occupational Health and Safety Act, makes the regulation in the attached Appendix, being the Coal Dust Regulation.

PETER LOUGHEED (Chairman)

APPENDIX

OCCUPATIONAL HEALTH AND SAFETY ACT

Coal Dust Regulation

1 In this regulation

(a) "exposed worker" means a worker whose duties require him, for at least 30 days in a 12-month period, to be in that part of a work site where coal mining or coal processing operations are conducted;

(b) "pulmonary function technician" means a person who has passed a pulmonary function technician course approved by the Director of Medical Services, or has been approved by the Director as having the equivalent of an approved pulmonary function technician course and who, in either case, has passed a requalification examination when requested by the Director to take one.

2 This regulation applies at work sites where coal is mined or processed in underground coal mines and surface coal mines, including ore beneficiation, coal preparation and load-out facilities.

3(1) Pursuant to an order under section 26 of the Act, a code of practice specifying safe working procedures respecting the control of coal dust shall include the work practice controls to be used to suppress airborne coal dust at each major source of dust generation.

(2) The employer shall ensure that copies of the code of practice and copies of this regulation are readily available to each worker.

4(1) Subject to section 8, the employer of a worker who becomes an exposed worker shall ensure that the worker undergoes or has undergone a medical assessment within 60 days before or after he becomes an exposed worker and not later than every 24 months after the date of that assessment and not less frequently than every 24 months thereafter, for as long as that worker is employed by the employer.

(2) If, on the coming into force of this regulation a worker is being examined in accordance with the Provincial Board of Health Regulations Respecting the Protection of Persons from Fibrosis of the Lungs (Division 25), (Alta. Reg. 375/71), the employer shall ensure that the worker undergoes a medical assessment not later than 24 months after his last examination under that regulation and not less frequently than every 24 months thereafter, for as long as that worker is employed by the employer.

5 A medical assessment consists of

(a) a P.A. chest x-ray of the worker on a 35 cm by 43 cm film,
(b) a pulmonary function test, including the spirogram,

forced air expiratory volume in 1.0 second (FEV$_1$) and forced vital capacity (FVC), all conducted by a pulmonary function technician,

(c) an assessment of the worker's ability to wear a respiratory protective device, and

(d) a written history specifying the worker's

(i) occupational exposure to dust,

(ii) respiratory symptoms including dyspnoea, cough, sputum production, wheezing or chest tightness,

(iii) incidence of chronic bronchitis, emphysema, asthma or other chronic lung disease, and

(iv) smoking history, including an estimation of duration and amount smoked.

6(1) The employer shall bear the cost of providing medical assessments under this regulation.

(2) A person who administers an x-ray or pulmonary function test or prepares an assessment or written history under section 5 shall, within 30 days thereafter, forward to the Director of Medical Services the x-ray film, the results of the pulmonary function test including the spirogram, a report of the assessment under section 5(c) or a copy of the written history, as the case may be.

7 A worker referred to in section 4 shall participate in a medical assessment provided pursuant to this regulation.

8 If a worker is already undergoing medical assessment pursuant to section 6 of the Silica Regulation (Alta. Reg. 9/82) or pursuant to section 6 of the Asbestos Regulation (Alta. Reg. 7/82) then sections 4 to 7 of this regulation do not apply to that worker.

9 Coal Workers' Pneumoconiosis is designated as a notifiable disease for the purpose of section 17 of the Act.

10(1) Section 2 of the Silica Regulation (Alta. Reg. 9/82) is repealed.

(2) The Provincial Board of Health Regulations Respecting the Protection of Persons from Fibrosis of the Lungs (Division 25), (Alta. Reg. 375/71) are repealed.

TABLE 2 OF SCHEDULE 1

OCCUPATIONAL EXPOSURE LIMITS FOR DUSTS

	8 hours Occupational Exposure Limit Respirable Mass mg/m³	Total Mass mg/m³
Silica (SiO_2)		
Amorphous	2	5
Cristobalite	0.05	0.15
Fused silica	0.1	0.3
Quartz	0.1	0.3
Silica flour	0.05	0.15
Tridymite	0.05	0.15
Tripoli	0.1	0.3
Aluminum oxide (Al_2O_3)	5	10
Calcium carbonate	5	10
Calcium silicate	5	10
Cellulose (paper fiber)	5	10
Coal dust	see footnote below	
Diatomaceous earth (natural)	2	5
Emery	5	10
Grain dust	-	4
Graphite (natural)	2.5	5
Graphite (synthetic)	5	10
Gypsum	5	10
Kaolin	5	10
Limestone	5	10
Marble	5	10
Magnesite	5	10
Mica	3	6
Nuisance particulate	5	10
Pentaerythritol	5	10
Perlite	5	10
Plaster of Paris	5	10
Portland cement	5	10
Rouge	5	10
Silicon	5	10
Silicon carbide	5	10
Soapstone	3	6
Starch	5	10
Sucrose	5	10
Talc (non-asbestiform)	2	4
Tin oxide	5	10
Titanium dioxide.	5	10
Zinc stearate	5	10
Zinc oxide dust	5	10

FOOTNOTE:

For coal dust,

(i) the 8 hour Occupational Exposure Limit is 2 mg/m³ (respirable mass) average for all operations in the mine;

(ii) the 8 hour Occupational Exposure Limit is 4 mg/m³ (respirable mass) in high risk operations.

TABLE 3 OF SCHEDULE 1

OCCUPATIONAL EXPOSURE LIMITS FOR FIBRES

	8 Hour OEL f/cm³	15 Minute OEL f/cm³	8 Hour OEL Total Mass mg/m³
Chrysotile	0.5	2.5	-
Amosite	0.2	1.0	-
Crocidolite	0.2	1.0	-
Tremolite	0.2	1.0	-
Talc (Fibrous)	2.0	10.0	-
Fibrous Glass	1.0	-	-
Mineral Wool	1.0	-	-
Refractory Ceramic Fibre	0.5	-	-

AR 393/88 Sched.1:303/92

Triphenyl amine	---	5	---	10	---	---
Triphenyl phosphate	---	3	---	6	---	---
Tungsten & Compounds, (as W)						
Soluble	---	1	---	3	---	---
Insoluble	---	5	---	10	---	---
Turpentine	100	560	150	840	---	---
Uranium (natural) soluble & insoluble compounds (as U)	---	0.2	---	0.6	---	---
Valeraldehyde	50	175	75	265	---	---
Vanadium, as $V_2 0_5$						
Respirable dust and fume	---	0.05	---	0.15	---	---
Vinyl acetate	10	35	20	70	---	---
Vinyl benzene (Styrene) - Skin	50	213	100	426	---	---
Vinyl bromide (Schedule 2)	5	22	10	44	---	---
Vinyl chloride (Chloroethylene) (Schedule 2)	2	5.2	10	26	---	---
Vinyl cyanide (Acrylonitrile) - Skin	2	4.3	4	8.6	---	---
Vinyl cyclohexene dioxide (Schedule 2)	10	57	15	86	---	---
Vinylidene chloride (1,1-Dichloroethylene)	5	20	10	40	---	---
Vinyl toluene	50	242	100	483	---	---
VM & P Naphtha	300	1350	400	1800	---	---
Warfarin	---	0.1	---	0.3	---	---
Welding fumes (total particulate)	---	5	---	10	---	---
Wood dust, nonallergenic	---	5	---	10	---	---

Wood dust, allergenic	---	2.5	---	5	---	---
Xylene (o-,m-,p-isomers) - Skin	100	434	150	652	---	---
m-Xylene alpha, alpha'-diamine	---	---	---	---	---	0.1
Xylidine (Dimethylaminobenzene) - Skin	5	25	10	50	---	---
Yttrium	---	1	---	3	---	---
Zinc chloride fume	---	1	---	2	---	---
Zinc chromate (as Cr) (Schedule 2)	---	0.05	---	0.15	---	---
Zinc oxide fume	---	5	---	10	---	---
Zirconium compounds (as Zr)	---	5	---	10	---	---

TABLE 2
OF
SCHEDULE 1

OCCUPATIONAL EXPOSURE LIMITS FOR DUSTS

	8 hour Occupational Exposure Limit	
	Respirable Mass mg/m³	Total Mass mg/m³
Silica (SiO_2)		
Amorphous	2	5
Cristobalite	0.05	0.15
Fused Silica	0.1	0.3
Quartz	0.1	0.3
Silica Flour	0.05	0.15
Tridymite	0.05	0.15
Tripoli	0.1	0.3
Aluminum oxide ($Al_2 0_3$)	5	10

Asbestos (Schedule 2)	see footnote (a)	
Calcium carbonate	5	10
Calcium silicate	5	10
Cellulose (paper fiber)	5	10
Coal dust	see footnote (b)	
Diatomaceous earth (natural)	2	5
Emery	5	10
Fibrous Glass	see footnote (c)	
Grain dust	--	4
Graphite (natural)	2.5	5
Graphite (Synthetic)	5	10
Gypsum	5	10
Kaolin	5	10
Limestone	5	10
Marble	5	10
Magnesite	5	10
Mica	3	6
Mineral Wool Fibre	see footnote (c)	
Nuisance Particulate	5	10
Pentaerythritol	5	10
Perlite	5	10
Plaster of Paris	5	10
Portland Cement	5	10
Rouge	5	10
Silicon	5	10
Silicon carbide	5	10
Soapstone	3	6
Starch	5	10
Sucrose	5	10

Talc (fibrous)	see footnote (d)	
Talc (nonasbestiform)	2	4
Tin oxide	5	10
Titanium dioxide	5	10
Zinc stearate	5	10
Zinc oxide dust	5	10

Footnotes:

(a) asbestos

(i) for asbestos fibre, except crocidolite, amosite and tremolite, the 8 hour Occupational Exposure Limit is 0.5 fibres greater than 5 micrometers in length per cm^3 of air; the 15 minute Occupational Exposure Limit is 2.5 fibres greater than 5 micrometers in length per cm^3 of air;

(ii) for crocidolite, amosite and tremolite fibre, the 8 hour Occupational Exposure Limit is 0.2 fibres greater than 5 micrometers in length per cm^3 of air;

the 15 minute Occupational Exposure Limit is 1 fibre greater than 5 micrometers in length per cm^3 of air;

(b) coal dust

(i) the 8 hour Occupational Exposure Limit is 2 mg/m^3 (respirable mass) average for all operations in the mine;

(ii) the 8 hour Occupational Exposure Limit is 4 mg/m^3 (respirable mass) in high risk operations;

(c) fibrous glass or mineral wool fibre

(i) for fibrous glass or mineral wool fibre, the 8 hour Occupational Exposure Limit is 3 fibres per cm^3 of air;

(ii) fibres included in this count are those having a diameter equal to or less than 3.5 micrometers and a length equal to or greater than 10 micrometers;

(iii) the 8 hour Occupational Exposure Limit is 5 mg/m^3 (total mass);

(d) talc (fibrous)

(i) for fibrous talc, the 8 hour Occupational Exposure Limit is 2 fibres greater than 5 micrometers in length per cm^3 of air;

(ii) the 15 minute Occupational Exposure Limit is 10 fibres greater than 5 micrometers in length per cm^3 of air.

PROVINCE OF ALBERTA

OCCUPATIONAL HEALTH AND SAFETY ACT

CHEMICAL HAZARDS REGULATION

Alberta Regulation 393/88 with amendments, up to and including Alberta Regulation 169/97

Consolidated January 21, 1998

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Part 3
Asbestos

Definitions and application 35
Hazardous work sites and occupations 36
Employer's general duties 37
Restricted areas 38
Re-usable protective clothing 39
Instructions for workers 40
Health assessments 41
Notification of certain new projects 43
Notifiable diseases 44

Part 4
Coal Dust

Definitions and application 45
Hazardous work sites and occupations 46
Employer's general duties 47
Instructions for workers 48
Health assessments 49
Notifiable diseases 51

Part 5
Silica

Definitions and application 52
Hazardous work sites and occupations 53
Employer's general duties 54
Instructions for workers 55
Health assessments 56
Notifiable diseases 58

Part 6
General

Transitional 59
Repeals 60

Schedules

PART 1

PROVISIONS OF GENERAL APPLICATION

Definitions

1 In this Regulation,

(a) "Act" means the *Occupational Health and Safety Act;*

(b) "harmful substance" means a substance that by its nature, application or presence creates or could create a danger to the health or safety of any worker who is exposed to it;

(c) "joint work site health and safety committee" means the committee, if any, established at a work site pursuant to an order under section 25 of the Act;

(d) "Occupational Exposure Limit", in respect of a substance, means the Occupational Exposure Limit or Limits established by Schedule 1 for that substance;

(e) "pulmonary function technician" means a person who

(i) has passed, or has been approved by a Director of Medical Services as having done the equivalent of passing, a pulmonary function technician course approved by a Director of Medical Services, and

(ii) if so required by a Director of Medical Services, has passed a requalification examination approved by such a Director.

AR 393/88 s1;303/92

Exposure to substances by inhalation

2(1) An employer shall ensure that each worker's exposure by inhalation to any substance listed in Schedule 1

(a) is kept as low as is reasonably practicable, and

(b) does not exceed its Occupational Exposure Limit.

(2) If no Occupational Exposure Limit has been established for a harmful substance present at a work site, an employer shall ensure that all steps are taken to keep each worker's exposure to that harmful substance as low as is reasonably practicable.

AR 393/88 s2

Exposure to 2 or more substances

3(1) An employer shall take all reasonable steps to ensure that where a worker is exposed to more than one substance listed in Schedule 1 in the same shift and where the toxicological effects of those substances are additive, the value of D in the formula

$$D = \frac{C_1}{T_1} + \frac{C_2}{T_2} + \dots \frac{C_n}{T_n}$$

does not exceed 1, where $C_1, C_2, \dots C_n$ refer to the actual airborne concentrations of contaminants 1, 2, ... n, and $T_1, T_2, \dots T_n$ are their respective 8 hour Occupational Exposure Limit.

(2) In calculating the formula contained in subsection (1), the employer shall express the numerators and the denominators of the fractions in the same units.

AR 393/88 s3

Disclosure of valid exemption

30 An employer who receives notice of a decision under the federal Information Review Act that his claim or a portion of his claim for exemption from a requirement to disclose information in respect of a controlled product on a material safety data sheet or a label is valid shall, during a period beginning not more than 30 days after the final disposition of the claim and ending on the last day of the exemption period, in respect of the sale or importation into Canada of the controlled product or any controlled product having the same product identifier, disclose on the material safety data sheet and, where applicable, on the label of the controlled product or the container in which the controlled product is packaged

(a) a statement that an exemption has been granted,

(b) the date of the decision granting the exemption, and

(c) the registry number assigned to the claim under the federal Information Review Act.

AR 393/88 s30

Officials' duty of confidentiality

31(1) Where an official working under the authority of the Act obtains information from the Commission under paragraph 46(2)(e) of the federal Information Review Act, he shall keep that information confidential and shall not disclose it to any person except for the purposes of the administration or enforcement of the Act and this Part.

(2) Any person to whom information is disclosed pursuant to this section shall keep the information confidential except for the purposes of the administration or enforcement of the Act and this Part.

(3) Subsections (1) and (2) apply notwithstanding any other law.

AR 393/88 s31;303/92

Medical emergencies

32(1) An employer shall provide such information respecting any controlled product, including confidential business information exempted from disclosure under section 28, as is in his possession to a medical professional who requests information on the controlled product for the purpose of rendering medical treatment to a person in an emergency.

(2) A person to whom confidential business information exempted from disclosure under section 28 is provided by an employer pursuant to subsection (1) shall not disclose the information to any other person except so far as may be necessary for the purpose of rendering medical treatment to a person in an emergency.

(3) Any person to whom confidential business information is disclosed under subsection (2) shall keep the information confidential.

AR 393/88 s32

Prohibition against disclosure of confidential business information

33(1) No person shall use or disclose information protected as confidential business information under this Regulation except as provided by sections 31 and 32.

(2) Subsection (1) does not apply to the person who has claimed an exemption for the confidential business information under section 28 or a person acting with that person's permission in respect of a use or disclosure.

AR 393/88 s33

Disclosure of source of toxicological data

34 Subject to the federal Information Review Act, an employer who manufactures a controlled product shall, at the request of

(a) an officer,

(b) any concerned worker,

(c) the joint work site health and safety committee, or

(d) in the absence of a joint work site health and safety committee, a representative of concerned workers,

disclose as quickly as possible under the circumstances the sources of any toxicological data used in preparing the material safety data sheet pursuant to section 26.

AR 393/88 s34

PART 3

ASBESTOS

Definitions and application

35(1) In this Part,

(a) "asbestos waste" means discarded materials from which there is a reasonable chance that asbestos might be released and become airborne, and includes disposable protective clothing that has been used in a restricted area;

(b) "exposed worker" means a worker who may reasonably be expected to work in a restricted area during at least 30 work days in a 12-month period;

(c) "restricted area" means an area of a work site where there is a reasonable chance of the concentration of airborne asbestos being at least 50% of the 8 hour Occupational Exposure Limit.

(2) Nothing in this Part relieves a person from any duty that he has under Part 1.

AR 303/92 s12

Hazardous work sites and occupations

36(1) For the purposes of the Act, each restricted area is designated as a hazardous work site.

(2) Where a worker works with asbestos or is engaged in the removal or abatement of asbestos or in the demolition of buildings or equipment containing asbestos, for the purposes of the Act, his occupation is designated as a hazardous occupation.

AR 303/92 s12

Employer's general duties

37 An employer shall

(a) take appropriate measures to minimize the release of asbestos into the air,

(b) keep the work site clear of unnecessary accumulations of asbestos and waste materials containing asbestos,

(c) ensure that methods used to decontaminate the work area, workers and protective clothing prevent the generation of airborne asbestos as far as is reasonably practicable,

(d) ensure that containers of asbestos products and asbestos waste are labelled to indicate the presence of asbestos and its carcinogenic hazard, with a warning that the dust should not be inhaled,

(e) ensure that asbestos waste is kept, transported and disposed of in sealed containers that are impervious to asbestos and asbestos waste, and

(f) provide facilities adequate to ensure that workers' street clothing is not contaminated by asbestos.

AR 303/92 s12

Restricted areas

38(1) An employer shall

(a) ensure that no one enters a restricted area except persons authorized by him or by law to do so,

(b) post in a conspicuous place at the entrances to and on the periphery of each restricted area signs that clearly indicate that

(i) asbestos is present in the area,

(ii) entry to the area is prohibited except to authorized persons, and

(iii) drinking, eating and smoking are prohibited in the area,

and keep them posted until certain that the area is no longer a restricted area,

(c) provide workers in the restricted area with protective clothing that prevents contamination of other clothing that is being worn,

(d) ensure that workers wear the protective clothing when they are in the restricted area, and

(e) ensure that workers are decontaminated before leaving the restricted area.

(2) A worker shall not leave a restricted area until he has decontaminated himself, unless emergency conditions preclude time for decontamination.

AR 303/92 s12

Re-usable protective clothing

39 An employer shall ensure that protective clothing that has been used in a restricted area and is to be re-used

(a) is laundered when necessary, and in any event not less frequently than after every 3 days of use, and

(b) where laundering is required by this section and until it is laundered, is stored and transported in sealed containers labelled to indicate the presence of asbestos and its carcinogenic hazard, with a warning that the dust should not be inhaled.

AR 303/92 s12

Instructions for workers

40 An employer shall ensure that workers who work with asbestos or are engaged in the removal or abatement of asbestos or in the demolition of buildings or equipment containing asbestos have successfully completed a course of instruction approved by a Director of Occupational Hygiene.

AR 303/92 s12

Health assessments

41(1) In this section, "health assessment" means the procedures described in subsection (8).

(2) An employer shall ensure at all times that he knows whether or not an exposed worker has received a health assessment within the past 2 years.

(3) If an exposed worker has received a health assessment from a previous employer within the past 2 years, the worker shall inform his present employer of the date or approximate date of that assessment at the earliest possible time.

(4) Within 30 days of a person's becoming an exposed worker, his employer shall ensure that the worker undergoes a health assessment, unless the worker has provided the employer with a written statement refusing it.

(5) An employer shall ensure that each exposed worker undergoes a health assessment at least every 2 years, unless the worker has provided the employer with a written statement refusing it.

(6) Where an exposed worker refuses a health assessment, he shall provide the employer with a written statement refusing it.

(7) Repealed AR 169/97 s2.

(8) The health assessment shall consist of

- (a) a 35 cm by 43 cm postero-anterior view chest x-ray, including a radiologist's report,
- (b) a spirogram, conducted by a pulmonary function technician, including determinations of
  - (i) forced expiratory volume in the first second, and
  - (ii) forced vital capacity,
- (c) a history covering
  - (i) the worker's occupational exposure to asbestos and to other industrial dusts and carcinogens, and any significant exposure to asbestos, dusts or carcinogens during recreational or hobby activities,
  - (ii) any significant symptoms that may be an indication of asbestosis or malignancy,
  - (iii) any past or present medical diagnoses of respiratory diseases,
  - (iv) the worker's smoking history,
  - (v) the dates of the chest x-ray and the spirogram, and
  - (vi) the identity of the worker and employer,

and

- (d) a written interpretation and explanation by a physician to the worker of the results of his health assessment, with particular reference to the worker's exposure to airborne asbestos.

(9) The employer shall bear the cost of providing health assessments and medical interpretations and explanations required by this Part.

(10) The employer shall ensure that the health assessment is, when practicable, performed during the normal hours of employment.

(11) When an exposed worker

- (a) has a health assessment during his hours of employment, or
- (b) spends time in going to or returning from that assessment during his hours of employment,

his employer shall not deduct any wages, salary or other remuneration or benefits in respect of the period so occupied.

(12) The person who has custody of the health assessment record shall ensure that no person, other than the worker, or the physician or nurse who conducts the health assessment, or the staff supervised by that physician or nurse, or any other person authorized by law to have such access, has access to an exposed worker's health assessment produced as a result of the application of this Part, unless

- (a) the record is in a form that does not identify the worker, or
- (b) the worker has given his written permission for access by another person.

(13) The physician who provides the written interpretation described in subsection (8)(d) to the worker must ensure that the records of the health assessment outlined in subsection (8) are maintained for a period of not less than 30 years.

AR 303/92 s12;169/97

42 Repealed AR 169/97 s3.

Notification of certain new projects

43 A person who is about to begin a new project involving the removal or abatement of asbestos or the demolition of a building or equipment containing asbestos shall notify a Director of Inspection of the project at least 72 hours before the project commences.

AR 303/92 s12

Notifiable diseases

44 Asbestosis, mesothelioma and asbestos induced lung, laryngeal and gastrointestinal cancer are designated as notifiable diseases for the purposes of section 17 of the Act.

AR 303/92 s12

PART 4

COAL DUST

Definitions and application

45(1) In this Part, "exposed worker" means a worker whose duties require him, for at least 30 days in a 12-month period, to be in that part of a work site where coal mining or coal processing operations are conducted.

(2) Nothing in this Part relieves a person from any duty that he has under Part 1.

AR 303/92 s12

Hazardous work sites and occupations

46 Where on a work site there is a reasonable possibility that a worker might be exposed to airborne coal dust, then, for the purposes of section 19 of the Act,

(a) that work site is a hazardous work site, and

(b) his occupation is a hazardous occupation.

AR 303/92 s12

Employer's general duties

47 An employer shall

(a) take appropriate measures to minimize the release of coal dust into the air, and

(b) keep his work site clear of unnecessary accumulations of coal dust.

AR 303/92 s12

Instructions for workers

48 An employer shall ensure that workers who may be exposed to airborne coal dust are instructed regarding the health hazards associated with exposure to coal dust.

AR 303/92 s12

Health assessments

49(1) In this section, "health assessment" means the procedures described in subsection (8).

(2) An employer shall ensure at all times that he knows whether or not an exposed worker has received a health assessment within the past 2 years.

(3) If an exposed worker has received a health assessment from a previous employer within the past 2 years, the worker shall inform his present employer of the date or approximate date of that assessment at the earliest possible time.

(4) Within 30 days of a person's becoming an exposed worker, his employer shall ensure that the worker undergoes a health assessment, unless the worker has provided the employer with a written statement refusing it.

(5) An employer shall ensure that each exposed worker undergoes a health assessment at least every 2 years, unless the worker has provided the employer with a written statement refusing it.

(6) Where an exposed worker refuses a health assessment, he shall provide the employer with a written statement refusing it.

(7) Repealed AR 169/96 s4.

(8) The health assessment shall consist of

(a) a 35 cm by 43 cm postero-anterior view chest x-ray, including a radiologist's report,

(b) a spirogram, conducted by a pulmonary function technician, including determinations of

  (i) forced expiratory volume in the first second, and

  (ii) forced vital capacity,

(c) a history covering

  (i) the worker's occupational exposure to coal dust and to other industrial dusts and carcinogens, and any significant exposure to coal dust and other dusts and carcinogens during recreational or hobby activities,

  (ii) any significant symptoms that may be an indication of a pneumoconiosis,

  (iii) any past or present medical diagnoses of respiratory diseases,

  (iv) the worker's smoking history,

  (v) the dates of the chest x-ray and the spirogram, and

  (vi) the identify of the worker and employer,

and

(d) a written interpretation and explanation by a physician to the worker of the results of his health assessment, with particular reference to the worker's exposure to airborne coal dust.

(9) The employer shall bear the cost of providing health assessments and medical interpretations and explanations required by this Part.

(10) The employer shall ensure that the health assessment is, when practicable, performed during the normal hours of employment.

(11) When an exposed worker

(a) has a health assessment during his hours of employment, or

(b) spends time in going to or returning from that assessment during his hours of employment,

his employer shall not deduct any wages, salary or other remuneration or benefits in respect of the period so occupied.

(12) The person who has custody of the health assessment record shall ensure that no person, other than the worker, or the physician or nurse who conducts the health assessment, or the staff supervised by that physician or nurse, or any other person authorized by law to have such access, has access to an exposed worker's health assessment produced as a result of the application of this Part, unless

(a) the record is in a form that does not identify the worker, or

(b) the worker has given his written permission for access by another person.

(13) The physician who provides the written interpretation described in subsection (8)(d) to the worker must ensure that the records of the health assessment outlined in subsection (8) are maintained for a period of not less than 30 years.

AR 303/92 s12;169/97

50 Repealed AR 169/97 s5.

Notifiable diseases

51 Coal worker's pneumoconiosis is designated as a notifiable disease for the purposes of section 17 of the Act.

AR 303/92 s12

PART 5

SILICA

Definitions and application

52(1) In this Part,

(a) "exposed worker" means a worker who may reasonably be expected to work in an area where there is a reasonable chance of the concentration of airborne silica being at least 50% of its 8 hour Occupational Exposure Limit, during at least 30 work days in a 12-month period;

(b) "silica" means crystalline silicon dioxide, including fused silica, silica flour, quartz, cristobalite, tridymite and tripoli.

(2) Nothing in this Part relieves a person from any duty that he has under Part 1.

AR 303/92 s12

Hazardous work sites and occupations

53 Where on a work site there is a reasonable possibility that a worker might be exposed to airborne silica, then, for the purposes of section 19 of the Act,

(a) that work site is a hazardous work site, and

(b) his occupation is a hazardous occupation.

AR 303/92 s12

Employer's general duties

54 An employer shall

(a) take appropriate measures to minimize the release of silica dust into the air, and

(b) keep his work site clear of unnecessary accumulations of silica dust.

AR 303/92 s12

Instructions for workers

55 An employer shall ensure that workers who may be exposed to airborne silica are instructed regarding the health hazards associated with exposure to silica.

AR 303/92 s12

Health assessments

56(1) In this section, "health assessment" means the procedures described in subsection (8).

(2) An employer shall ensure at all times that he knows whether or not an exposed worker has received a health assessment within the past 2 years.

(3) If an exposed worker has received a health assessment from a previous employer within the past 2 years, the worker shall inform his present employer of the date or approximate date of that assessment at the earliest possible time.

(4) Within 30 days of a person's becoming an exposed worker, his employer shall ensure that the worker undergoes a health assessment, unless the worker has provided the employer with a written statement refusing it.

(5) An employer shall ensure that each exposed worker undergoes a health assessment at least every 2 years, unless the worker has provided the employer with a written statement refusing it.

(6) Where an exposed worker refuses a health assessment, he shall provide the employer with a written statement refusing it.

(7) Repealed AR 169/96 s6.

(8) The health assessment shall consist of

- (a) a 35 cm by 43 cm postero-anterior view chest x-ray, including a radiologist's report,
- (b) a spirogram, conducted by a pulmonary function technician, including determinations of
  - (i) forced expiratory volume in the first second, and
  - (ii) forced vital capacity,
- (c) a history covering
  - (i) the worker's occupational exposure to silica and to other industrial dusts and carcinogens, and any significant exposure to silica and other dusts and carcinogens during recreational or hobby activities,
  - (ii) any significant symptoms that may be an indication of silicosis,
  - (iii) any past or present medical diagnoses of respiratory diseases,
  - (iv) the worker's smoking history,
  - (v) the dates of the chest x-ray and the spirogram, and
  - (vi) the identity of the worker and employer,

and

- (d) a written interpretation and explanation by a physician to the worker of the results of his health assessment, with particular reference to the worker's exposure to airborne silica.

(9) The employer shall bear the cost of providing health assessments and medical interpretations and explanations required by this Part.

(10) The employer shall ensure that the health assessment is, when practicable, performed during the normal hours of employment.

(11) When an exposed worker

- (a) has a health assessment during his hours of employment, or
- (b) spends time in going to or returning from that assessment during his hours of employment,

his employer shall not deduct any wages, salary or other remuneration or benefits in respect of the period so occupied.

(12) The person who has custody of the health assessment record shall ensure that no person, other than the worker, or the physician or nurse who conducts the health assessment, or the staff supervised by that physician or nurse, or any other person authorized by law to have such access, has access to an exposed worker's health assessment produced as a result of the application of this Part, unless

- (a) the record is in a form that does not identify the worker, or
- (b) the worker has given his written permission for access by another person.

(13) The physician who provides the written interpretation described in subsection (8)(d) to the worker must ensure that the records of the health assessment outlined in subsection (8) are maintained for a period of not less than 30 years.

AR 303/92 s12;169/97

57 Repealed AR 169/97 s7.

Notifiable diseases

58 Silicosis is designated as a notifiable disease for the purposes of section 17 of the Act.

AR 303/92 s12

PART 6

GENERAL

Transitional **59(1)** *If an exposed worker has not received a health assessment between February 1, 1991 and January 31, 1993, his employer shall ensure that he undergoes a health assessment before February 28, 1993 unless the worker has provided the employer with a written statement refusing it, and sections 41, 49 and 56 apply in relation to such a health assessment.*

(2) In subsection (1), "exposed worker" has the meanings ascribed to it by sections 35(1), 45(1) and 52(1) respectively and "health assessment" has the meanings ascribed to it by sections 41(1), 49(1) and 56(1) respectively.

AR 393/88 s35;303/92

Repeals **60** *The following are repealed:*

(a) the Asbestos Regulation (Alta. Reg. 7/82);

(b) the Silica Regulation (Alta. Reg 9/82);

(c) the Coal Dust Regulation (Alta. Reg. 243/83).

AR 393/88 s36;303/92

SCHEDULE 1

Interpretation **1**(1) In this Schedule,

(a) "ceiling Occupational Exposure Limit" means the maximum concentration at any point in time of an airborne substance listed in Table 1 of this Schedule;

(b) "coal dust" means a dust

(i) resulting from the mining, transportation or processing of coal,

(ii) of carboniferous or mixed mineralogical composition, and

(iii) containing 10% or less of free silica calculated by weight;

(c) "8 hour Occupational Exposure Limit" means the time-weighted average concentration of an airborne substance listed in this Schedule for an 8 hour period;

(d) "fibre" means a particulate material having

(i) a diameter equal to or less than 3 micrometres,

(ii) a length equal to or greater than 5 micrometres, and

(iii) an aspect (length/diameter) ratio equal to or greater than 3:1;

(e) "15 minute Occupational Exposure Limit" means the time-weighted average concentration of an airborne substance listed in this Schedule for a 15 minute period;

(f) "mg/m³" means milligrams of substance per cubic metre of air measured at standard conditions of 25 degrees C and 101.3 kiloPascals;

(g) "nuisance particulate" means a particulate material that does not produce a documented toxic effect and is not otherwise specified in this Schedule;

(h) "ppm" means parts of vapour or gas by volume per million parts of contaminated air by volume;

(i) "R" means registered trade mark;

(j) "respirable mass" means that mass of the total airborne particulate that can be inhaled and deposited in the lower respiratory tract.

(2) When it appears in conjunction with a substance in this Schedule, "Skin" means that the substance can be absorbed through the intact skin.

(3) For the purposes of calculating the 15 minute time-weighted average concentration for determining compliance with the 15 minute Occupational Exposure Limit listed in this Schedule, the sampling period must

(a) start at the moment when the airborne concentration exceeds the absolute value listed in that Table as the 15 minute Occupational Exposure Limit, and

(b) continue for the next 15 minutes.

(4) Each 15 minute period referred to in subsection (3) must be followed by a period of at least 60 minutes during which the airborne concentration of the substance is at or below the absolute value listed in Table 1 of this Schedule as the 8 hour Occupational Exposure Limit.

Appendix 4

Forms Provided for Use in the Alberta Fibrosis Program

CONSENT FORM

DIRECTOR OF MEDICAL SERVICES
OCCUPATIONAL HEALTH & SAFETY DIVISION
WORKERS' HEALTH, SAFETY & COMPENSATION
3RD FLOOR, OXBRIDGE PLACE
9820-106 STREET
EDMONTON, ALBERTA
T5K 2J6

THE OCCUPATIONAL HEALTH & SAFETY DIVISION CAN TRANSMIT INFORMATION REGARDING MY CHEST X-RAYS AND BREATHING TESTS TO THE DOCTOR OR PERSONS NAMED BELOW:

NAME OF DOCTOR/PERSON: ______________________________
(PLEASE PRINT)

ADDRESS: ______________________________
____

NAME OF DOCTOR/PERSON: ______________________________
(PLEASE PRINT)

ADDRESS: ______________________________
____

☐ I GIVE MY CONSENT

☐ I DO NOT GIVE MY CONSENT

______________________________ DATE

______________________________ SIGNATURE

WHSB1044 - BCA/DEC 80

FP-5

Alberta
WORKERS' HEALTH SAFETY & COMPENSATION

EMPLOYMENT AND EXPOSURE HISTORY

MEDICAL SERVICES BRANCH
OCCUPATIONAL HEALTH & SAFETY DIVISION
WORKERS' HEALTH, SAFETY & COMPENSATION
3RD FLOOR, OXBRIDGE PLACE, 9820-106 ST
EDMONTON, ALBERTA T5K 2J6

NAME: ______________________________
(SURNAME) (GIVEN NAMES)

____
ADDRESS PHONE NUMBER

OCCUPATIONAL HISTORY (LIST PREVIOUS JOBS IN CHRONOLOGICAL ORDER.)

PREVIOUS JOB TITLE	TYPE OF WORK	DURATION OF EMPLOYMENT	EMPLOYE

(PLEASE COMPLETE ON BACK OF PAGE IF NECESSARY)

HAVE YOU EVER SUFFERED FROM AN OCCUPATIONAL DISEASE OR INJURY AND SUBMITTE
A CLAIM FOR WORKERS' COMPENSATION PAYMENTS? ☐ *YES* ☐ *N*
IF SO PLEASE SPECIFY.

____
____
____
____

EXPOSURE HISTORY (HAVE YOU EVER WORKED WITH OR BEEN EXPOSED TO:)

	YES	NO	YEARS FROM	YEARS TO	TOTAL NO. YEARS
1. ASBESTOS DUST.........	☐	☐	*19*___	*19*___	___
2. COAL DUST.............	☐	☐	*19*___	*19*___	___
3. FIBERGLASS............	☐	☐	*19*___	*19*___	___
4. SILICA DUST........... (SAND BLASTING, QUARRY ETC.)	☐	☐	*19*___	*19*___	___
5. OTHER DUST EXPOSURES..	☐	☐	*19*___	*19*___	___

ADDITIONAL INFORMATION:

____
____
____

____
SIGNATURE

WHS81043

____
DATE

FP-

MEDICAL FINDINGS FP-8

RE:
HOME ADDRESS:
SOCIAL INSURANCE NUMBER:
DATE OF BIRTH:

PULMONARY FUNCTION TEST RESULTS

Date of Examination:

F.E.V.$_1$ - Observed =
-Predicted =
% =
F.V.C. - Observed =
-Predicted =
% =

Senior Medical Consultant
Medical Services Branch

RADIOLOGY REPORT

Date of X-ray:
Date of Interpretation:
Report:

Radiological Medical Consultant.

81 01

Alberta
WORKERS' HEALTH SAFETY & COMPENSATION

MEDICAL SERVICES BRANCH
OCCUPATIONAL HEALTH & SAFETY DIVISIO
WORKERS' HEALTH, SAFETY & COMPENSATI
3RD FLOOR, OXBRIDGE PLACE, 9620-106
EDMONTON, ALBERTA T5K 2.

REQUEST FOR REPEAT XRAY

1. FILE NUMBER ☐

2. SOCIAL INSURANCE NUMBER ☐☐☐ ☐☐☐ ☐☐☐

3. NAME OF WORKER ______________________________

4. DATE OF XRAY ☐☐☐☐☐☐
Y Y M M D D

DEAR SIR:

THE XRAY ON THE ABOVE WORKER IS UNREADABLE FOR THE FOLLOWING REASON(

☐ POSITION ☐ TOO LIGHT

☐ ARTIFACT ☐ TOO DARK

☐ OTHER SPECIFY ______________________

A REPEAT XRAY IS REQUESTED (AT YOUR EARLIEST CONVENIENCE)

____
RADIOLOGICAL MEDICAL CONSULTANT

OCCUPATIONAL HEALTH AND SAFETY
MEDICAL SERVICES
FIBROSIS CODING SHEET

FILE NUMBER	1 1	2
EMPLOYEE NAME		9
SOCIAL INSURANCE NUMBER		33
AHCIP		42
DATE OF BIRTH (YYMMDD:YY-YEAR,MM-MONTH,DD-DAY)		53
SEX (M-MALE,F-FEMALE,X-UNKNOWN)		59
EMPLOYER ACCOUNT NUMBER		60
MSB CODE		66
INDUSTRY		69
OCCUPATION	1-MINERS 2-ASBESTOS/INSULATORS 3-DUST AND OTHERS	74
XRAYS (YYO:YY-YEAR,O-OUTCOME)		75
OUTCOME	1-XRAY/NO OUTCOME 2-NORMAL 3-ABNORMAL 4-UNACCEPTABLE	84 93
PFT (YYO;YY-YEAR,O-OUTCOME)		99
OUTCOME	1-PFT/NO OUTCOME 2-NORMAL 3-ABNORMAL 4-UNACCEPTABLE	108 117

WMS8100-

40

FP-D

Alberta
WORKERS' HEALTH SAFETY & COMPENSATION

NOTE: PLEASE RECORD YOUR INTERPRETATION OF
A SINGLE FILM BY PLACING AN "X" IN THE
APPROPRIATE BOXES ON THIS FORM

RADIOLOGIST CODING SHE

SOCIAL INSURANCE NO ☐☐☐☐☐☐☐☐☐

NAME: ____________________

FILE NO. ☐

1. DATE OF X-RAY ☐☐☐☐☐☐

2. IS FILM COMPLETELY NORMAL? YES ☐ NO ☐

3. FILM QUALITY 1 2 3 U/R

IF NOT GRADE GIVE REASON

☐ POSITION ☐ TOO LIGH
☐ ARTIFACT ☐ TOO DARK

4. SMALL OPACITIES

B. PROFUSION

0/-	0/0	0/1
1/0	1/1	1/2
2/1	2/2	2/3
3/2	3/3	3/4

C. ZONES

R	L
☐	☐
☐	☐
☐	☐

A. SHAPE/SIZE

PRIMARY		SECONDARY	
P	S	P	S
Q	T	Q	T
R	U	R	U

5. LARGE OPACITIES (PNEUMOCONIOSIS)

SIZE A B C

6. PLEURAL THICKENING

A. DIAPHRAGM (PLAQUE) SITE R L

B. COSTOPHRENIC ANGLE SITE R L

7. PLEURAL THICKENING .... CHEST WALL

A. CIRCUMSCRIBED (PLAQUE)			B. DIFFUSE		
SITE	R	L	SITE	R	L
IN PROFILE I. WIDTH	A B C	A B C	IN PROFILE I. WIDTH	A B C	A B C
II. EXTENT	1 2 3	1 2 3	II. EXTENT	1 2 3	1 2 3
EN FACE III. EXTENT	1 2 3	1 2 3	EN FACE III. EXTENT	1 2 3	1 2 3

8. PLEURAL CALCIFICATION SITE R EXTENT L EXTENT

A. DIAPHRAGM....	1 2 3	A. DIAPHRAGM....	1 2 3
B. WALL.........	1 2 3	B. WALL.........	1 2 3
C. OTHER SITES..	1 2 3	C. OTHER SITES..	1 2 3

9. OTHER SYMBOLS (OBLIGATORY)

AX AZ BU CA CN CO CP CR CT CV DI EF EM ES FR HI HO HY ID IH KL NM OT PC

PI PX RP SC SN ST TB ☐ ☐

DATE OF READING ☐☐☐☐☐☐

FILM READERS INITIAL

10. IS THERE PROGESSION FROM LAST FILM YES NO

11. OTHER COMMENTS ____________________

SHOULD WORKER SEE DOCTOR BECAUSE OF COMMENTS IN THIS SECTION YES NO

WHS510-2 - BCA/DEC 83

FP-C

Alberta
WORKERS' HEALTH SAFETY & COMPENSATION

MEDICAL SERVICES BRANCH
OCCUPATIONAL HEALTH & SAFETY DIVISION
WORKERS' HEALTH, SAFETY & COMPENSATIC
3RD FLOOR, OXBRIDGE PLACE, 9820-106 S
EDMONTON, ALBERTA TSK 2.

1. **IDENTITY OF COMPANY:** NAME OF COMPANY: ______________________
ADDRESS: ______________________
CONTACT PERSON AT COMPANY: ______________________

2. **IDENTITY OF WORKER:** NAME: ______________________ (SURNAME) (FIRST NAME, INITIALS)
BIRTHDATE: ______________ (DAY / MONTH / YEAR) S.I.N.: ______________
HEIGHT: ________ SEX: M ___ F ___ PAYROLL NO.: ________
OCCUPATION: ______________________

3. **SMOKING STATUS:**
SMOKER ________ NON-SMOKER ________ EX-SMOKER ________

4. **PULMONARY FUNCTION TEST RESULTS:** DATE OF EXAMINATION: ______________ (DAY / MONTH / YEAR)

	OBSERVED	PREDICTED	% OBSERVED PREDICTED	PERFORMED BY:
FEV	________	________	________	______________ (NAME)
FEC	________	________	________	______________ (AGENCY)

5. **CHEST X-RAY:** DATE OF X-RAY: ______________ (DAY / MONTH / YEAR)
RADIOLOGY REPORT: ____ ENCLOSED ____ NOT ENCLOSED

6. **DECLARATION:**
THIS FILE WAS PREPARED AND FORWARDED TO THE MEDICAL SERVICES BRANCH
ON ______________ (DAY / MONTH / YEAR) ______________________ (SIGNATURE OF COMPANY OFFICIAL)
______________________ (TITLE OF COMPANY OFFICIAL)

7. **REMARKS:** ______________________
____

SENIOR MEDICAL CONSULTANT SUMMARY SHEET

NAME OF INDIVIDUAL: ____
(SURNAME) (FIRST NAME, INITIALS)

PULMONARY FUNCTION TEST: *NORMAL* _____ *ABNORMAL* _____

CHEST X-RAY: *NORMAL* _____ *ABNORMAL* _____

IMPRESSION: _____ *NORMAL*

_____ *SUSPECT OCCUPATIONAL DISEASE*

SPECIFY: ______________________________

_____ *SUSPECT NON-OCCUPATIONAL DISEASE*

SPECIFY: ______________________________

CERTIFICATE ISSUED:

_____ *YES* *IF YES: DATE ISSUED:* ______________

_____ *NO* *IF NO: REPEAT X-RAY* ____ *REPEAT PFT* ___

OTHER: ____________________

REFERRAL TO DOCTOR:

_____ *YES*

_____ *NO*

FURTHER COMMENTS: ____

____

____

____

____

WHSB1045 - BCA/DEC 80

FP-B

INDEX CARD

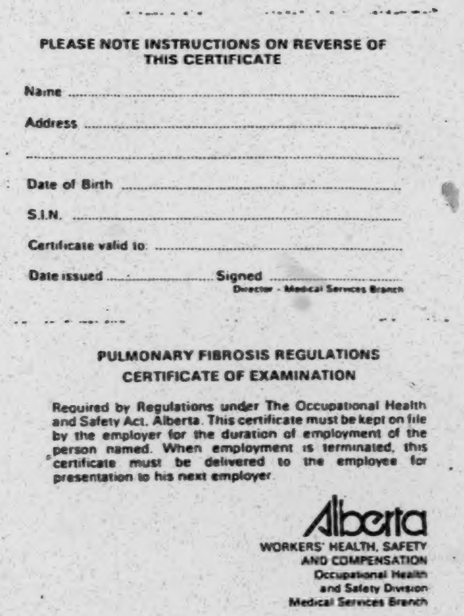
Index card kept on each worker in the program.

NAME	HEIGHT	D.O.B.	S.I.N.

X-Ray	P.F.T.		Cert.

CERTIFICATE

Certificate issued after chest x-ray and pulmonary function test of satisfactory quality have been evaluated.



PLEASE NOTE INSTRUCTIONS ON REVERSE OF THIS CERTIFICATE

Name ....

Address ....

....

Date of Birth ....

S.I.N. ....

Certificate valid to: ....

Date issued .... Signed ....
Director - Medical Services Branch

PULMONARY FIBROSIS REGULATIONS
CERTIFICATE OF EXAMINATION

Required by Regulations under The Occupational Health and Safety Act. Alberta. This certificate must be kept on file by the employer for the duration of employment of the person named. When employment is terminated, this certificate must be delivered to the employee for presentation to his next employer.

Alberta
WORKERS' HEALTH, SAFETY
AND COMPENSATION
Occupational Health
and Safety Division
Medical Services Branch

HEALTH ASSESSMENT / WORKER REGISTRY

EMPLOYER:

NAME

ADDRESS

CITY

LOCATION

CONTACT PERSON

HEALTH ASSESSMENT ☐ DONE ☐ REFUSED

PHYSICIAN WHO PROVIDED HEALTH ASSESSMENT:

EMPLOYEE:

SURNAME FIRST NAME INITIALS

ADDRESS

CITY

SEX ☐ M ☐ F BIRTH DATE (D/M/Y) HEIGHT (CM)

SURNAME AT BIRTH PROVINCE OF BIRTH

AHCIP SIN

DURING THE YEAR, WERE YOU EXPOSED TO AIRBORNE (CHECK ONLY ONE)
☐ COAL ☐ SILICA ☐ ASBESTOS ☐ OTHER DUST ☐ MAN MADE MINERAL FIBRE

JOB DESCRIPTION

OCCUPATIONAL EXPOSURE/HISTORY

CHECK (X) IF YOU WORK OR HAVE EVER WORKED AT OR WITH:
☐ COALMINE ☐ QUARRY ☐ FOUNDRY ☐ ASBESTOS ☐ SILICA ☐ OTHER DUSTY JOB

OCCUPATION	PERIOD (YRS)	EXPOSURE TO OCCUPATIONAL HAZARD

CHEST SYMPTOMS

CHECK (X) IF YOU SUFFER FROM OR HAVE EVER SUFFERED FROM:
☐ FREQUENT COLDS ☐ DYSPNEA ☐ CHRONIC COUGH ☐ SPUTUM PRODUCTION ☐ WHEEZING ☐ CHEST TIGHTNESS ☐ CHEST PAIN ☐ EASY FATIGUE

PAST ILLNESS

CHECK (X) IF YOU SUFFERED FROM ANY OF THE FOLLOWING ILLNESSES:
☐ HEART TROUBLE ☐ BRONCHITIS ☐ PNEUMONIA ☐ PLEURISY ☐ TUBERCULOSIS ☐ ASTHMA ☐ OTHER CHEST TROUBLE ☐ HAY FEVER ☐ EMPHYSEMA

SMOKING HISTORY

DO YOU SMOKE NOW? ☐ YES ☐ NO IF YES, HOW MANY? (# PER DAY) ____________

HAVE YOU EVER SMOKED? ☐ YES ☐ NO NUMBER OF YEARS ____________ HOW LONG SINCE YOU QUIT? ____________ YEARS

FITNESS TO WEAR RESPIRATORY PROTECTIVE EQUIPMENT

IS THIS WORKER FIT TO WEAR ALL TYPES OF RESPIRATORY PROTECTIVE EQUIPMENT? ☐ YES ☐ NO

IF NO, SPECIFY TYPES WORKER IS FIT TO WEAR ____________________________

CHEST X-RAY

RADIOLOGIST REPORT INCLUDED: ☐ YES ☐ NO DATE OF X-RAY ____________ DAY MONTH YEAR

PULMONARY FUNCTION TEST

DATE OF PFT ____________ DAY MONTH YEAR

	Observed	Predicted	% Observed Predicted	Performed by
FEV1				NAME
FVC				AGENCY

FOR OFFICE USE ONLY:

PFT AND XRAY OUTCOME

XRAY OUTCOME ☐ PFT OUTCOME ☐ 1–NORMAL 2–ABNORMAL 3–UNACCEPTABLE 4–NO TEST RECEIVED

ICD ____________ ILO ☐ ☐ DISEASE ☐ 1–NOT STATED 2–OCCUPATIONAL 3–NON-OCCUPATIONAL

[illegible]-Oct. 28, [illegible]

FIBROSIS PROGRAM FORM